The Journal of Bone and Joint Surgery

The Official Publication of

The American Orthopaedic Association
The British Orthopaedic Association
The American Academy of Orthopaedic Surgeons
The Australian Orthopaedic Association
The Canadian Orthopaedic Association

JANUARY 1950

American Volume

Vol. 32-A, No. 1

8 THE FENWAY, BOSTON 15, MASSACHUSETTS, U. S. A.
BRITISH OFFICE:
LUDGATE HOUSE, FLEET STREET, E.C. 4, ENGLAND

The Journal of Bone and Joint Surgery

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PUBLISHED IN TWO VOLUMES

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THE JOURNAL OF BONE AND JOINT SURGERY

American Office:
8 The Fenway, Boston 15, Massachusetts, U.S.A.
Assistant Editor: **Florence L. Daland**

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Medical

VOL. 32-A, NO. 1 JANUARY 1950

The Journal of
Bone and Joint Surgery

American Volume

STABILIZING OPERATIONS ON THE FOOT

A Study of the Indications, Techniques Used, and End Results *

BY ROBERT LEE PATTERSON, JR., M.D., FRANK F. PARRISH, M.D., AND ELWOOD N. HATHAWAY, M.D., NEW YORK, N. Y.

From the Orthopaedic Service of the Hospital for Special Surgery, New York City

At The Hospital for Special Surgery in New York, over 1,300 operations for stabilization of the foot were performed between the years 1935 and 1947, inclusive. During the past year, 305 of these procedures were studied, as carried out on 233 patients. No patient was examined less than one year after operation. Since other members of our Staff have reported the results of astragalectomy [15], of posterior bone block [16], and of stabilization of the medial border of the foot for congenital flat-foot [14], these findings have been omitted from the present study. The patients who returned for re-examination were those who had had one or more of the following eight procedures:

1. Hoke stabilization
2. Brewster countersinking operation
3. Triple arthrodesis
4. Lambrinudi stabilization
5. Panarthrodesis
6. Subtalar arthrodesis
7. Primary wedge osteotomy
8. Revision procedures for failure of a primary bone procedure.

These operations were carried out by sixty-six surgeons. Their results are subject to all of the variables which occur when different surgeons perform operations, especially in such an institution as The Hospital for Special Surgery, where the teaching of residents undergoing postgraduate training in orthopaedic surgery is a primary objective.

Indications for Operation

Any operative procedure for stabilization is intended to correct deformity or instability or to relieve pain which greatly handicaps a patient in normal stance or gait. In numerous instances, the reconstruction has been performed to permit the fitting of a brace on an otherwise useless extremity, in order that ambulation might be started.

A study of the records in the 1,300 cases mentioned shows that operations have been done for a number of different conditions (Table I).

Discussion of Technique of Operation

The patient is placed on the table on his side, with the extremity to be operated upon uppermost (Fig. 1). The hip and knee are each flexed about 30 degrees. The medial border

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 25, 1949.

TABLE I

ETIOLOGICAL INDICATIONS FOR FOOT STABILIZATION

1. Flaccid Paralysis
   - Poliomyelitis
   - Spina bifida
   - Friedreich's ataxia
   - Polyneuritis
   - Charcot-Marie-Tooth atrophy
   - Muscle dystrophy
   - Postencephalitis
   - Arnold-Chiari syndrome
   - Pelizaeus-Merzbacher disease
2. Spastic Paralysis
   - Infantile cerebral palsy (spastic and other types)
   - Hemiplegia and paraplegia (due to trauma and other causes)
3. Congenital Anomalies
   - Recurrent and uncorrected club-foot
   - Arthrogryposis multiplex congenita
   - Idiopathic claw-foot (questionable)
4. Painful Flat-Foot
   - Spastic flat-foot
   - Congenital weakness of the foot
   - Rigid flat-foot (calcaneonavicular coalition and other congenital anomalies)
5. Arthritis of the Foot
   - Rheumatoid arthritis
   - Osteo-arthritis or post-traumatic arthritis
6. Trauma
   - Dislocation or fracture-dislocation (old)
   - Malposition of fractures in mid-tarsal or hind-foot region
7. Infection
   - Septic
   - Tuberculous
   - Charcot joint (possibly due to syphilis)

of the foot is placed on a sandbag, which serves to make it parallel with the table and at the same time offers support when the joints are being resected. Care is taken to avoid

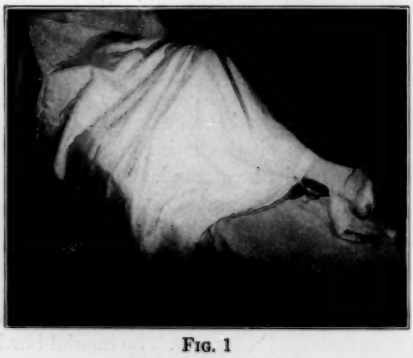

FIG. 1

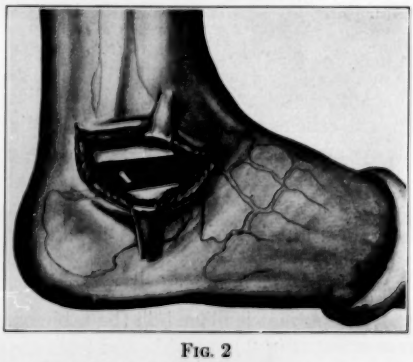

FIG. 2

Fig. 1: Position of the patient on the operating table for foot stabilization.
Fig. 2: Tendon of flexor hallucis longus is seen on medial side of foot.

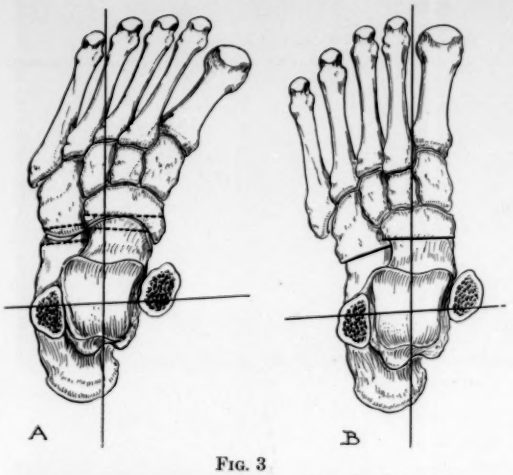

FIG. 3

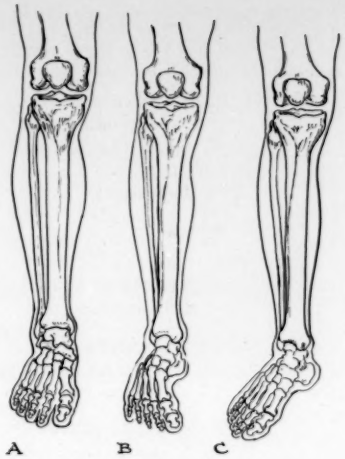

FIG. 4

Fig. 3, *A:* Failure to remove navicular "beak" may prevent lateral displacement of adducted fore part of the foot.

Fig. 3, *B:* Shows correction of deformity with good alignment, following resection.

Fig. 4: Alignment of foot.

A: Normal foot, ankle, and knee alignment without tibial torsion.

B: Incorrect: Foot is aligned with knee, not ankle, in the presence of lateral tibial torsion.

C: Correct: Foot is aligned with ankle joint in the presence of lateral tibial torsion.

injury to the sural nerve and the branches of the superficial peroneal nerve. After the origins of the short extensor muscles have been dissected, the sinus tarsi has been cleaned out, and the fibulocalcaneal ligament has been cut, the talonavicular, the calcaneocuboid, and the subtalar joints can be seen clearly.

The joint surfaces are resected with an osteotome rather than being cleaned with a curette, to ensure removal of all cartilage. After the talocalcaneal joint has been resected, the tendon of the flexor hallucis longus comes into view on the medial side of the foot (Fig. 2). The posterior tibial nerve and artery lie beyond this, so that great care must be given to the soft tissue in this area. There is often a "beak" or "hook" on the navicular (Fig. 3,*A*) which projects backward along the medial side of the head and neck of the talus. This is taken off flush with the main body of the navicular, through a separate incision if necessary, as it often prevents lateral displacement of the fore part of the foot (Fig. 3,*B*). At this point the foot is in three distinct "pieces",—namely, the fore part, the calcaneus, and the remnant of the talus firmly fixed within the ankle mortise. Each of these "pieces" can be moved in any direction independently of the others. The foot is then lined up properly with the ankle mortise. *It is never aligned with the knee* (Fig. 4).

If there is tibial torsion, it may be necessary to rotate the calcaneus internally or externally beneath the talus and, of course, the fore part of the foot is lined with the hind portion. After a wedge has been taken or after simple resection of joints, close contact between all resected surfaces is imperative. If necessary, temporary fixation with Steinmann pins or Kirschner wires should be resorted to for maintenance of contact, alignment, and stability. Once this has been effected, suture of the fibulocalcaneal ligament and one suture through the bone at the calcaneocuboid joint will help to maintain the corrected position of the foot until the wound has been closed and application of plaster has been completed.

Postoperative Management

The patient is taken from the operating theater with the extremity elevated in a special rack (Fig. 5-A), to avoid any period of dependency. The elevation is maintained

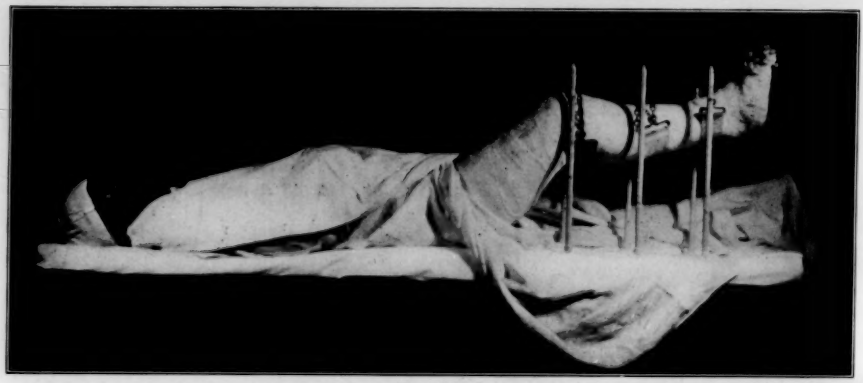

FIG. 5-A

Postoperative elevation of extremity in "astragalectomy" rack.

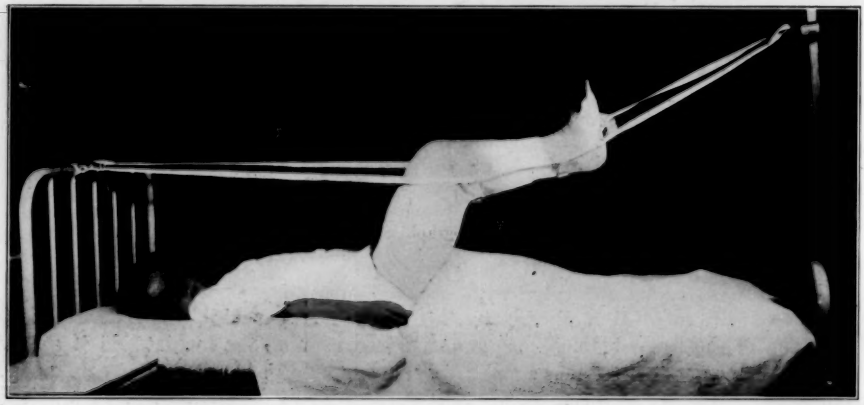

FIG. 5-B

Elevation by means of a sling.

by means of a sling for the first week (Fig. 5-B), after which short periods of dependency are allowed. These are gradually increased in duration and frequency. At the end of three weeks, the sutures are removed and a below-the-knee plaster is applied, after which roentgenograms are taken. Ambulation with crutches is permitted at this time. A walking heel is placed on the plaster at four to six weeks following operation, and weight-bearing is allowed. The plaster is again removed at the end of twelve weeks. When fusion is secure, as proved by roentgenographic examination and only by this means, the leg is wrapped with an adhesive elastic bandage and progressive weight-bearing is started with the use of crutches.

Roentgenograms

Three routine views (Figs. 6-A, 6-B, and 6-C) are taken, both before and after operation. These consist of a dorsoplantar projection of the foot, an anteroposterior projection of the ankle, and a lateral projection of the foot and ankle.

Method of Study and Grading

Every patient reported in this series had roentgenograms and photographs on his

FIG. 6-A

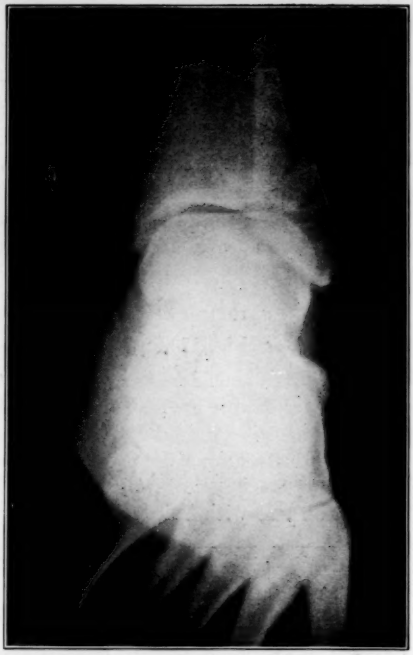

FIG. 6-B

Routine roentgenographic projections used:
Fig. 6-A: Dorsoplantar view of foot. Fig. 6-B: Anteroposterior view of ankle joint.

FIG. 6-C

Lateral view of foot and ankle.

return to the Hospital and was personally examined by one of the authors. It was found that 269 primary bone operations and 36 revision procedures had been performed on 233 patients.

In the evaluation of end results, both the anatomical and functional aspects must be taken into consideration, as stressed by Hallgrímsson in his excellent monograph. We have established our standards on the basis of an ideal foot (Figs. 7-A and 7-B), but have graded the outcome of the cases as good (Figs. 8-A and 8-B), fair (Figs. 9-A and 9-B), or failure (Figs. 10-A and 10-B). The end result has not been down-graded in a case of poliomyelitis because of foot-drop, or in a cerebral spastic because of a shuffling gait (Table II).

DESCRIPTION OF FINDINGS IN THE EIGHT OPERATIVE PROCEDURES

Hoke Arthrodesis (Fig. 11)

The authors considered an operation to be a Hoke arthrodesis [7] when the head and neck of the talus were excised, with a portion later reinserted and the talocalcaneal joint resected; in all cases except five the calcaneocuboid joint was included.

This operation was done on twenty-seven patients with poliomyelitis, on sixteen with some type of spastic paralysis, on five with congenital flat-foot, on five with congenital club-foot, on two with Friedreich's ataxia, and on one with rheumatoid arthritis. Every deformity and combination of deformities is represented in this series. The patients were operated upon at an average age of 13.3 years, the youngest being seven and the oldest thirty-seven, and were examined on an average of 5.9 years after operation. At the time of the arthrodesis, five supplemental procedures were employed, consisting of two transplants of the extensor

FIG. 7-A

FIG. 7-B

Excellent result.

Fig. 7-A: Postoperative photographs following stabilization of left foot.

Fig. 7-B: Postoperative roentgenograms of same foot.

hallucis longus to the neck of the first metatarsal, two transplants of the peroneal tendons, and one plantar fasciotomy. At the time of the follow-up study, three of these patients complained of some degree of pain, seven of varying amounts of fatigue, and four of some instability. The total period of immobilization averaged 11.6 weeks.

Correction of the deformity was good in twenty-seven cases, fair in twenty-two, and poor in seven. Of twenty-four feet showing varying amounts of fixed equinus before operation, five had some residual difficulty. Of seventeen patients who had drop-foot before surgery, thirteen had such deformities postoperatively. In none of the seventeen patients were tendons transplanted into the dorsum of the foot. Valgus deformity appeared in twenty-six preceding the bone procedure, and was well corrected in all but seven. Varus, noted twenty-one times before surgery, remained in twelve. Cavus, present in nineteen before surgery, existed in eight at follow-up. Of four patients having calcaneus deformity before operation, incomplete correction was present in two.

Pseudarthrosis occurred in only four patients, each time at the talonavicular joint. In three of these, postoperative roentgenograms showed the resected surfaces to be in poor contact. The average period of immobilization for these four patients was 12.5 weeks. Only one of the four had pain. The healing was reported as poor roentgenographically in all four instances, when the plaster had been removed; but, in spite of this warning, immobilization was discontinued. There was one peripheral-nerve injury in the series, and one patient had chronic phlebitis which has subsequently been relieved by lumbar sympathetic blocks.

The end results of the fifty-six Hoke arthrodeses were as follows:

	Number	*Per cent.*
Good	33	58.9
Fair	16	28.6
Failure	7	12.5

FIG. 8-A

FIG. 8-B

Good result.

Fig. 8-A: Postoperative photographs following triple arthrodesis of left foot.
Fig. 8-B: Postoperative roentgenograms of same foot.

TABLE II

CRITERIA USED FOR EVALUATION OF END RESULTS

Ideal Result
Fusion of all joints
No pain or instability
Heel well aligned in the lateral plane
Foot aligned with ankle joint
Foot hits ground flatly in heel-toe gait
Dorsiflexion limited sufficiently to give push-off
Plantar flexion limited sufficiently to avoid steppage or drop-foot gait
Good Result
Fusion of all joints
Minimal pain or instability
No callosities on plantar surface of foot
Foot aligned with ankle joint
Optimal correction of deformity (equinus, varus, valgus, calcaneus, cavus, or combinations of these abnormalities)
Heel-toe gait
Fair Result
Fusion of at least two joints (including talocalcaneal articulation)
Definite improvement in pain and stability
Mild callosities or none at all
Moderate correction of deformity
Failure (one or more of the following)
Failure of fusion of talocalcaneal joint (other joints may or may not be fused)
Persistence of marked pain or instability
Marked plantar callosities
Definite undercorrection, overcorrection, or recurrence of deformity

All seven failures, without exception, were due to recurrence or undercorrection of the deformity. In four of these, roentgenograms immediately after operation, while the leg was in plaster, showed failure of correction of the deformity. A fifth patient had loss of position in plaster or at the time the plaster was changed, and pseudarthrosis at the talonavicular joint; his foot had not been aligned properly with the ankle. The sixth patient had a recurrence of deformity due to muscle imbalance, and such severe ankle instability that a

FIG. 9-A

FIG. 9-B

Fair result.

Fig. 9-A: Photograph eleven years following Brewster countersinking operation on right foot.
Fig. 9-B: Painless pseudarthrosis of the talonavicular joint resulted, the only cause for this rating.

panarthrodesis was later required. The seventh case was listed as recurrence of deformity due to poor ankle alignment.

The forty-five patients upon whom fifty-six Hoke arthrodeses were performed had fewer complaints at re-examination than had patients treated by any other operative method. The percentage of pseudarthrosis was far smaller, yet the average age of patients at operation was 13.3 years,—the youngest of any of the groups examined. This analysis indicates that the Hoke stabilization can be adapted successfully to the correction of any combination of foot deformities. The greatest number of failures occurred after varus deformities and drop-foot. Residual varus most frequently represents timidity on the part of the surgeon in taking adequate lateral wedges, failure to maintain immobilization until the arthrodesis has been consolidated, improper alignment of the foot with the ankle joint, or a combination of all of these factors. This is entirely a technical failure which is not peculiar to the Hoke stabilization, but is common to all stabilizations reported here. This procedure by itself cannot be relied upon to correct drop-foot, but if adequate muscle power is available which can be transposed to the dorsum of the foot, the prognosis for alleviation of this deformity is excellent.

FIG. 10-A

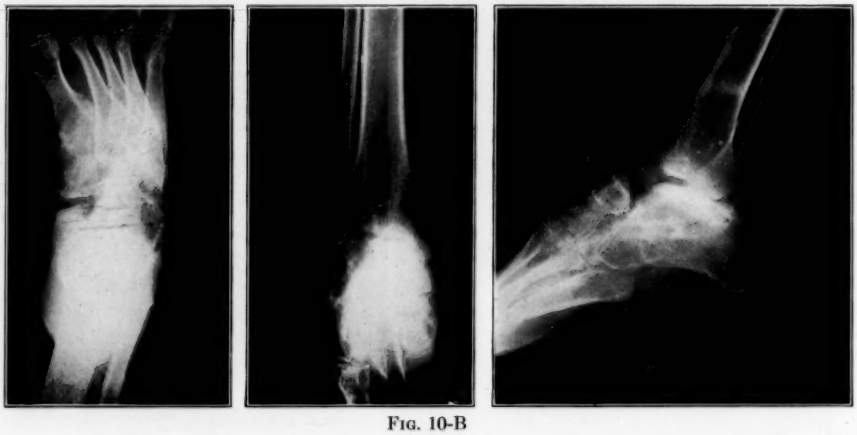

FIG. 10-B

Failure.

Fig. 10-A: Photographs taken after Lambrinudi stabilization show causes for failure rating.
Fig. 10-B: Follow-up roentgenograms showing deformity and pseudarthrosis of talonavicular joint, which was painful.

Brewster Countersinking Operation (Fig. 12)

This procedure[1] was carried out for deformity resulting from poliomyelitis in nine instances and from spastic paralysis in one. It was performed nine times for equinus deformity and once for a calcaneocavus flail foot. The average age at operation was 19.5 years, and the patients were seen at an average of 5.7 years after surgery. At the time of

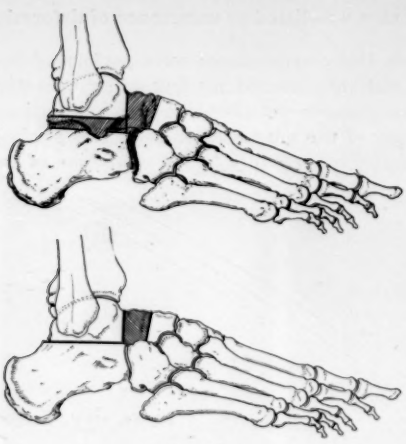

FIG. 11
Hoke arthrodesis.

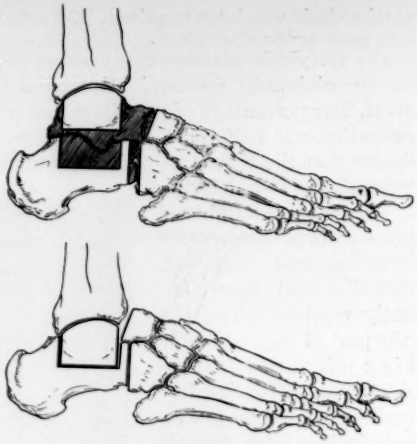

FIG. 12
Brewster countersinking operation.

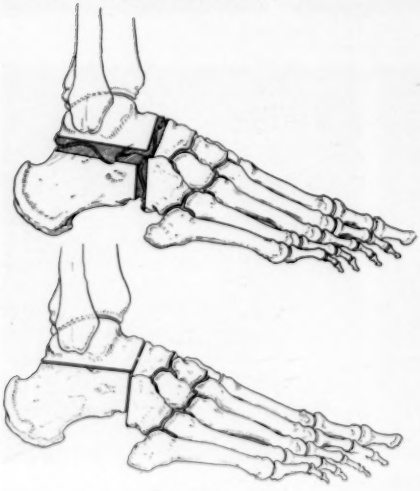

FIG. 13
Triple arthrodesis.

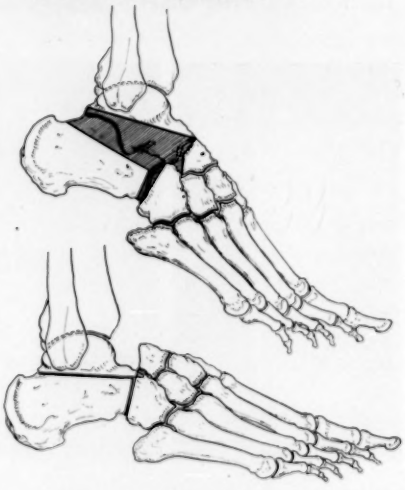

FIG. 14
Lambrinudi stabilization.

re-examination three patients had ankle pain, two complained of ankle instability, and four of foot fatigue.

The total period of immobilization in plaster averaged 12.2 weeks. Three of the operations were accompanied by tendon transplantation; two of the transplants were rated as active at the follow-up examination.

Five patients showed good correction of their deformity, four partial correction, and one a poor correction. In each instance of undercorrection, drop-foot was the deformity which remained. The foot seemed well displaced backward in each of the ten cases. In two cases which had tendon transplantation to the dorsum of the foot, there was no residual drop-foot.

Pseudarthrosis developed in two cases, each time at the talonavicular joint. A strong tendency was noted for the talus to be displaced out of the notch fashioned for it in the

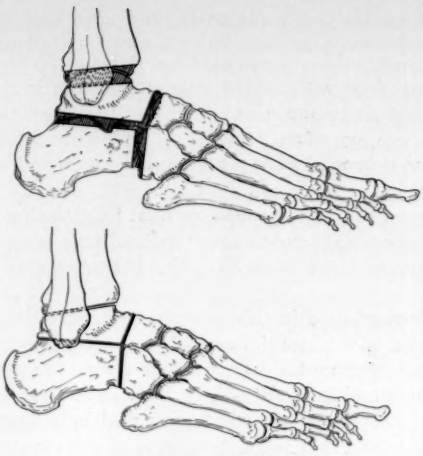

FIG. 15
Panarthrodesis.

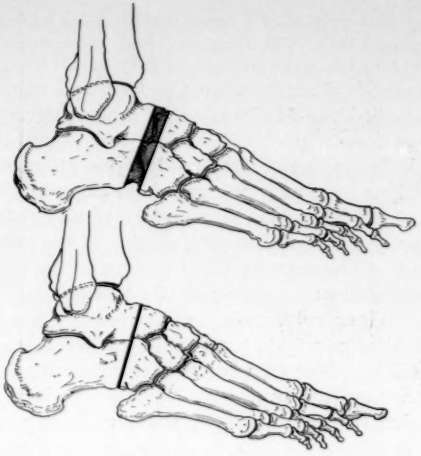

FIG. 16
Wedge osteotomy.

calcaneus, particularly during the first few days after operation.

The end results of ten Brewster countersinking operations were as follows:

	Number	*Per cent.*
Good	5	50
Fair	3	30
Failure	2	20

Of the failures, one, in a case of spastic paralysis, was due to pseudarthrosis at the talonavicular joint, accompanied by severe pain; the other was due to only partial correction of the deformity, accompanied by severe ankle instability.

From the study of this small group of patients, we have the impression that, technically, the Brewster countersinking procedure, as described in the literature, is difficult to carry out. There appears to be a tendency for displacement of the talus from the countersunk position and, unless adequate muscle power is available for transplantation to the dorsum of the foot, the operation cannot be relied upon to correct drop-foot.

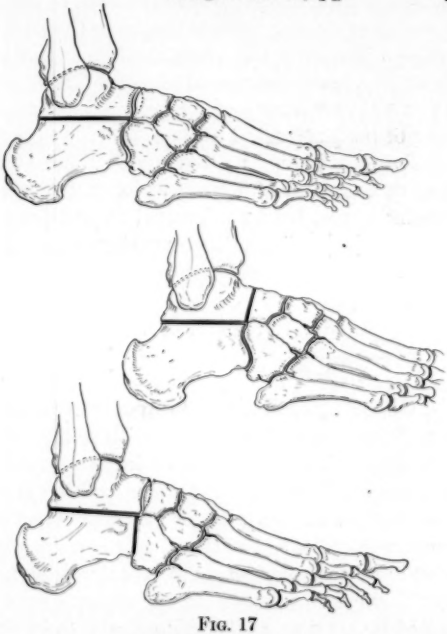

FIG. 17
Subtalar arthrodesis.

Triple Arthrodesis (Fig. 13)

Resection of the cartilaginous surfaces of the talonavicular, calcaneocuboid, and talocalcaneal joints, with correction of deformity in an architectural manner, was considered by the authors to be a triple arthrodesis [11]. This stabilization was done for poliomyelitis in forty-seven instances, for Friedreich's ataxia in twelve, for spastic paralysis in ten,

for deformities of a congenital nature in fourteen, for trauma in three, and once each for spina bifida, old fracture, and infectious arthritis. Equinocavovarus and equinovarus deformities were present in fifty-seven feet, simple valgus in twenty-one, flail foot in four, rocker-bottom deformity in two, and calcaneal foot with added valgus or varus in five others. Drop-foot was noted preoperatively in twenty-seven of the cases. The operation was done when the patients were at an average age of 16.7 years. At follow-up study, which averaged 5.4 years after operation, some degree of pain was found in nineteen feet, fatigue in twenty-one, and instability in four.

Additional surgery performed at the time of the bone procedure was: lengthening of the tendo achillis in five, section of the posterior tibial tendon in seven, and transplantation of the anterior tibial tendon to the dorsum of the foot in five. The period of plaster immobilization averaged 12.7 weeks.

Good correction of the deformity was accomplished in thirty-seven feet, partial correction in forty-two, and poor correction in seven. In the remaining cases, the operation was performed for pain and not for deformity. The component of varus, either alone or in combination with one of the other deformities, remained in twenty-three feet. Drop-foot still existed in eighteen feet, although in one of these the peronaeus longus had been transplanted to the dorsum of the foot, and another had had transplantation of the extensor hallucis longus to the neck of the first metatarsal. Of the nine successful recoveries, four had transplantation of the peronaeus longus to the dorsum of the foot and one had transplantation of the extensor hallucis longus to the neck of the first metatarsal. The remaining four regained muscle power when the long-standing equinus was overcome and the foot was placed in a good anatomical position. Twice rocker-bottom deformity occurred as a result of forceful dorsiflexion of the foot at the time of operation, when fixed equinus deformity had not been corrected.

Pseudarthrosis occurred twelve times at the talonavicular joint, and in one of these cases there was also failure of fusion at the talocalcaneal joint. In six of the twelve cases of pseudarthrosis, pain was present to some degree; in two, it was of sufficient severity to result in the grading of the operation as a failure.

The final grading for the eighty-nine cases of triple arthrodesis was as follows:

	Number	*Per cent.*
Good	48	53.9
Fair	27	30.3
Failure	14	15.7

Of the failures, nine had undercorrection of the deformity immediately after operation. Recurrence of deformity with failure of fusion at the talocalcaneal joint and poor ankle-foot alignment appeared in two patients. One had marked overcorrection of the deformity with muscle imbalance and too short a period of immobilization. Another patient had failure of fusion of the talonavicular and talocalcaneal joints, accompanied by severe pain. The last patient had depression in a plantar direction of the first metatarsal head in an equinus foot, with an extremely painful callosity under the head of that bone.

Triple arthrodesis is, in the authors' experience, the most commonly used stabilizing procedure. It does not lend itself well to backward displacement of the foot, as the head and neck of the talus are left intact. It is not so adaptable to the correction of a fixed equinus as the Lambrinudi procedure, nor was it used in the correction of so many types and combinations of deformities as was the Hoke stabilization.

Lambrinudi Stabilization (Fig. 14)

This operation [2, 6, 8, 9] was done fifty-five times. In thirty-five instances it was carried out for poliomyelitis, in four for Friedreich's ataxia, in four for some other type of neurogenic foot disorder, and in one instance for drop-foot in which the etiology was not clear. The deformities present before surgery were as follows: fixed equinus, forty; drop-foot,

nineteen; varus, thirty-one; valgus, eighteen; cavus, twenty-four; and calcaneus, eight. The most common single deformity for which the operation was chosen was fixed equinus. The patients were examined on an average of 5.1 years after operation. At that time, thirteen complained of some degree of pain in the foot, nine of fatigue, and four of instability.

The operations were performed when the patients were at an average age of 14.4 years. Immobilization averaged thirteen weeks.

Thirty of these patients showed good correction of their deformity, twenty showed partial correction, and five showed poor correction. Only three patients had residual fixed equinus of the fore part of the foot and the hind part; three had fixed equinus of the fore part of the foot alone. Twelve patients were left with incomplete correction of varus of the fore and hind parts of the foot, three with only some varus of the fore part of the foot. Twelve still had drop-foot deformity. Of the patients with residual drop-foot, one had undergone transplantation of the extensor hallucis longus to the neck of the first metatarsal, and another transplantation of the peronaeus longus to the navicular. Of the seven patients who had had correction of their drop-foot, in four the peronaeus longus had been transplanted to the dorsum of the foot, and in three the deformity was overcome without tendon transplantation. These patients recovered because of a return of muscle tone when the foot was placed in a more anatomical position as a result of the bone procedure.

There were sixteen failures of fusion at the talonavicular joint, and one each at the talocalcaneal and calcaneocuboid joints. In six of the talonavicular-joint pseudarthroses, poor contact was evident immediately after operation, while the leg was in plaster. This was also true of the talocalcaneal failure. This group of patients, in whom non-union developed, had an average period of immobilization in plaster of twelve weeks. Of the six talonavicular joints in which contact was poor, immobilization averaged thirteen weeks. In five of the six, poor healing took place in the arthrodesis, as shown by roentgenograms when the plaster had been removed; this also occurred in the patient with failure of stabilization at the talocalcaneal joint. These facts show the need of contact of resected surfaces in arthrodesis and of further immobilization when roentgenograms show delayed or poor healing.

The final grading of the fifty-five Lambrinudi stabilizations was as follows:

	Number	*Per cent.*
Good	25	45.5
Fair	22	40.0
Failure	8	14.5

Of the failures, two were due to painful pseudarthrosis and six to residual deformity. Three of the last six showed definite undercorrection of the deformity immediately after operation, while the leg was in plaster. This was further exaggerated by poor ankle-foot alignment, too early immobilization in two cases, and muscle imbalance in a third. Of the three with deformity, one had poor ankle-foot alignment; mobilization was carried out too soon, and non-union developed at the talonavicular joint. The second had poor ankle-foot alignment and recurrence of varus; and the final case showed uncorrected drop-foot with painful callosities on both the medial and lateral aspects of the foot.

This analysis of fifty-five surgical end results shows that the Lambrinudi procedure is preferable for the correction of fixed equinus. This preference has been well justified, since the Lambrinudi operation far surpasses all other bone operations for correction of this deformity. It again emphasizes the inability of foot stabilization alone to correct drop-foot; the chances of the average patient with paralysis regaining enough muscle power to actively dorsiflex the foot are small unless lateral muscles for transplantation are available and can be used. The susceptibility of the talonavicular joint to non-union appears to be increased by this particular operation, as witnessed by 29 per cent. of pseudarthrosis in this series. Therefore, in such a procedure, the authors would like to stress the necessity for adequate contact of a large surface of bone at this operative area plus adequate roentgenographic evidence of secure healing before immobilization is discontinued.

TABLE III

END RESULTS IN 305 FOOT STABILIZATIONS

Procedure	No. of Operations	Result			Patient Evaluation		
		Good	Fair	Failure	Pleased	Dis-pleased	Question-able
Hoke stabilization	56	33	16	7	49	2	5
Brewster countersinking	10	5	3	2	7	3	
Triple arthrodesis	89	48	27	14	70	11	8
Lambrinudi stabilization	55	25	22	8	46	9	
Panarthrodesis	20	13	5	2	18	2	
Subtalar arthrodesis	16	6	5	5	11	1	4
Wedge osteotomy	23	10	5	8	19	4	
Revisions	36	17	10	9	32	4	
Total	305	157 (51.48%)	93 (30.49%)	55 (18.03%)	252 (82.6%)	36 (11.8%)	17 (5.6%)

Panarthrodesis (Fig. 15)

This operation [3, 5, 10, 12, 13] was performed twenty times, in each instance for poliomyelitis. One patient had had previous stabilization and posterior bone-block operation, complicated by avascular necrosis of the talus. Panarthrodesis was carried out for deformity or instability in all but this last case; here the operation was done for relief of pain. The deformity in the nineteen cases was dangle foot (flail foot), or drop-foot, combined with varying degrees of varus, valgus, cavus, or calcaneus deformity. The operation was done at an average age of 20.7 years, the follow-up at an average of 4.7 years after operation. Four patients complained of some ankle pain, one of tarsal pain after walking a long distance, and two of pain referable to the fifth metatarsal head. Some degree of ankle or foot fatigue was present in seven patients, but none complained of instability.

Seven of the operations were completed in one stage and thirteen in two stages, with several weeks or months elapsing between procedures. Total immobilization averaged 22.5 weeks. Pseudarthrosis developed in four patients, one having failure of fusion at two joints. Three of the non-unions occurred at the talonavicular joint and two at the ankle joint. In four of these resected joints, contact between the surfaces was poor, as shown by roent-

TABLE IV

RESULTS OF FOOT STABILIZATION IN POLIOMYELITIS

Type of Procedure	No. of Procedures	Average Length of Follow-up (*Years*)	Evaluation		
			Good	Fair	Failure
Subtalar arthrodesis	7	8.7	3	1	3
Wedge osteotomy	11	7.2	5	1	5
Brewster countersinking	9	5.6	5	3	1
Hoke stabilization	27	6.7	13	10	4
Lambrinudi stabilization	35	5.9	15	15	5
Triple arthrodesis	46	5.1	27	13	6
Panarthrodesis	20	4.7	13	5	2
Total	155	5.9	81 (83.2%)	48	26 (16.8%)

TABLE V

CORRECTION OF DEFORMITY IN POLIOMYELITIS

Deformity	Number	Per cent. of Correction
Drop-foot (including dangle foot or flail foot)	95	45.3
Equinus	76	86.9
Varus	75	45.3
Cavus	56	78.6
Valgus	43	88.4
Calcaneus	19	73.7

genograms taken immediately after operation, and it remained poor. In the fifth, the talonavicular joint, delayed union was noted when the patient's leg was removed from plaster. The average time of immobilization of these five joints was thirty-nine weeks. In three cases the pseudarthrosis has subsequently been repaired successfully, and the results are satisfactory. Six patients in the series had some type of wound difficulty. Sloughing occurred in three patients whose panarthrodesis had been done in one stage. In one case the sloughing was very extensive, requiring a pedicle graft from the abdomen, but a successful result was finally obtained. Three patients had fatigue fractures of the lower end of the tibia, at the site of the sliding graft.

In fourteen of the cases, optimal correction of the deformity was attained. Six showed partial correction; of this group, three had undercorrection of the varus and two others had added varus and adduction of the fore part of the foot.

The end results of the twenty panarthrodeses were as follows:

	Number	*Per cent.*
Good	13	65
Fair	5	25
Failure	2	10

Eighteen patients were pleased with the result. In both failures, residual varus deformity was present with painful pseudarthrosis at the ankle joint. One patient complained of the equinus position of the foot, but all of the rest were pleased with fusion in equinus.

Comparison of the one-stage and two-stage procedures suggests that, where there is any question of circulatory impairment, the two-stage procedure is safer. Of the four instances of wound sloughing, three occurred after the one-stage procedure. Three of the five pseudarthroses were also associated with the one-stage procedure; and in all, poor contact of the resected joint surfaces appeared immediately after operation. It is reasonable to assume that it is much more difficult to get accurate apposition of all raw bone surfaces in a four-joint arthrodesis than in a triple arthrodesis.

Wedge Osteotomy (Fig. 16)

This procedure implies an osteotomy in the foot at the site of greatest deformity, irrespective of bones or joints. This was done in eleven patients with poliomyelitis, six with club-foot, three with Friedreich's ataxia, and one each with spina bifida, idiopathic claw-foot, and arthrogryposis multiplex congenita. A review of the deformities revealed that some component of varus was present in twenty-one feet, cavus in nineteen, and equinus in sixteen.

The average age of the patients at the time of operation was 15.1 years. The period of immobilization in plaster averaged twelve weeks. At re-examination, five feet had some degree of pain, three in association with callosities on the plantar surface of the foot. Five had varying degrees of fatigue and one patient had instability.

Six of these patients had optimal correction of their deformity, fourteen had fair cor-

rection, and three had poor correction. Of twenty-one patients in whom varus was present before operation, thirteen had residual varus of the heel and thirteen had varus or adduction of the fore part of the foot. Of nineteen patients with cavus, the condition remained incompletely corrected in seven; and of the sixteen showing equinus before operation, six were left with some degree of this deformity. The causes of residual deformity were loss of position in one case, failure of fusion in five, insufficient bone removed from the proper place in fourteen, and muscle imbalance in three. Nine patients had good alignment of the foot with the ankle mortise; in six the alignment was fair, and in four it was poor.

Pseudarthrosis occurred after seven of the twenty-three operations. In four of these, there was poor contact of the raw bone surface at the point where the wedge had been removed. The total average period of immobilization in these seven cases was 10.3 weeks.

The end results of the twenty-three osteotomies were as follows:

	Number	*Per cent.*
Good	10	43.5
Fair	5	21.7
Failure	8	34.8

In the failures, each showed some residual degree of varus; four had poor ankle-foot alignment and painful pseudarthrosis.

The results of wedge osteotomies show a sharp increase in the percentage of failures as compared to the entire group of stabilizations. These results emphasize that heel varus cannot be corrected by a mid-tarsal wedge osteotomy, nor can proper ankle-foot alignment be obtained by this means. Of these patients, pseudarthrosis developed in 30 per cent. This fact, plus the previously mentioned faults of the operation, indicates that the method is a poor one and should not be used in place of any of the three-joint procedures.

Subtalar Arthrodesis (Fig. 17)

Although the authors considered in this series that a subtalar arthrodesis included only the talocalcaneal joint, often the talonavicular or the calcaneocuboid joint was also fused, but never both.

Seven of the patients gave a history of poliomyelitis and three of fracture involving the talocalcaneal joint; one had rheumatoid arthritis; three had some type of neurogenic foot disorder; and two of the procedures were done in one patient who had bilateral rigid flat-foot with some arthritic changes. In six of the cases the operation was done for pain without deformity, in four for equinovarus, in three for valgus drop-foot, in one for varus drop-foot, and in one each for cavovarus and calcaneovalgus. Surgery was performed when the patients were at an average age of 23.1 years, and the patients were seen an average of 7.7 years after operation. The period of plaster immobilization averaged 10.8 weeks. Two

TABLE VI

RESULTS OF FOOT STABILIZATIONS IN SPASTIC PARALYSIS

Type of Procedure	No. of Procedures	Average Length of Follow-up (*Years*)	Evaluation		
			Good	Fair	Failure
Subtalar arthrodesis	2	6.8	1	1	0
Brewster countersinking	1	7.5	0	0	1
Hoke stabilization	15	4.7	10	2	3
Lambrinudi stabilization	10	4.3	4	4	2
Triple arthrodesis	10	5.3	4	4	2
Total	38	4.9	19 (78.9%)	11	8 (21.1%)

TABLE VII

CORRECTION OF DEFORMITY IN SPASTIC PARALYSIS

Deformity	Number	Per cent. of Correction
Equinus	19	94.7
Varus	18	77.7
Valgus	16	68.8
Cavus	11	72.7
Rocker bottom	8	75.0
Calcaneus	5	100.0

pseudarthroses occurred at the talonavicular joint and one at the talocalcaneal joint.

The end results of the sixteen subtalar arthrodeses were as follows:

	Number	*Per cent.*
Good	6	37.5
Fair	5	31.25
Failure	5	31.25

Of the five failures, two patients had marked undercorrection of their deformity immediately after operation and one, operated upon at the age of seven, had a recurrence of deformity due to muscle imbalance and insufficient immobilization. In all three of these patients, the principal component of the deformity was varus. In the fourth patient, also operated upon at seven years, pseudarthrosis of the talocalcaneal joint developed and the calcaneovalgus was converted into an equinovarus deformity. The fifth patient was a man of fifty, who had fusion of the talocalcaneal and calcaneocuboid joints following an old fracture of the calcaneus. He was improved, but still had marked pain.

FIG. 18-A

Preoperative photographs show Friedreich's ataxia of left foot.

The results in this group of cases emphasize the difficulty in correcting deformity unless a three-joint arthrodesis is done. They further point out the value of adequate immobilization and of delaying the operation until bone maturation has advanced satisfactorily. If we combine the totals of these arthrodeses with those of the group with wedge osteotomy, we have thirty-nine cases in which two-joint arthrodeses have been done. Thus the percentage of satisfactory results is much smaller than in the total group (Table III).

FIG. 18-B

Photographs of successful stabilization of left foot.

RESULTS ON THE BASIS OF ETIOLOGY

In 247 of the 269 primary stabilizations, the operation was performed for poliomyelitis, spastic paralysis, Friedreich's ataxia, congenital club-foot, or painful flat-foot.

Poliomyelitis

Panarthrodesis was used exclusively in poliomyelitis; 155 operations were performed for this disease (Table IV). The average age of the patients at the onset of paralysis was 3.2 years. The average time between contraction of the disease and stabilization was 12.4 years, the shortest interval being 3.5 years. Tendon transplantation, supplementing the arthrodesis or done at a later date, seemed of more value than a similar procedure done prior to the bone operation. This was notable in the correction of drop-foot and in the removal of a deforming muscle to avoid future recurrence of deformity.

Drop-Foot

The 45.3 per cent. of correction (Table V) includes treatment by panarthrodesis and tendon transplantation. No patient in this series had correction of this deformity without muscle transplantation or without regaining sufficient muscle tone to dorsiflex the foot actively. Fatigue was a complaint in sixteen cases.

Spastic Paralysis

The majority of these patients had had soft-part surgery before the stabilization was done (Table VI). Varus appeared more easily correctable in the patients with spastic paralysis than in those in any of the other four groups (Table VII). The removal of a deforming element by section of the posterior tibial tendon, carried out in half of the cases, may have been a factor.

FIG. 19-A

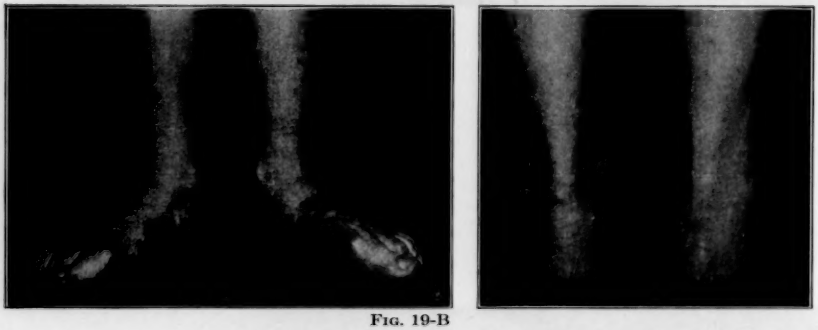

FIG. 19-B

Correction of painful flat-foot.
Fig. 19-A: Before stabilization. Fig. 19-B: After stabilization.

TABLE VIII

RESULTS OF FOOT STABILIZATIONS IN FRIEDREICH'S ATAXIA

Type of Procedure	No. of Procedures	Average Length of Follow-up (*Years*)	Evaluation		
			Good	Fair	Failure
Subtalar arthrodesis	1	5.7	0	0	1
Wedge osteotomy	3	5.9	1	2	0
Hoke stabilization	2	7.3	1	1	0
Lambrinudi stabilization	4	4.2	1	2	1
Triple arthrodesis	12	6.5	5	2	5
Total	22	6.0	8 (68.2%)	7	7 (31.8%)

TABLE IX

CORRECTION OF DEFORMITY IN FRIEDREICH'S ATAXIA

Deformity	Number	Per cent. of Correction
Cavus	21	52.4
Varus	21	57.1
Equinus	20	65.0

The failures were due equally to pain, pseudarthrosis, and residual deformity. In all other groups the principal cause of failure was residual deformity.

Friedreich's Ataxia

The average age at the onset of symptoms was 8.5 years. The patients were operated

TABLE X

RESULTS OF FOOT STABILIZATIONS IN CLUB-FOOT

Type of Procedure	No. of Procedures	Average Length of Follow-up (*Years*)	Evaluation		
			Good	Fair	Failure
Wedge osteotomy	6	6.4	3	2	1
Hoke stabilization	5	5.3	2	3	0
Lambrinudi stabilization	1	6.0	1	0	0
Triple arthrodesis	7	7.2	4	3	0
Total	19	6.4	10 (94.7%)	8	1 (5.3%)

TABLE XI

CORRECTION OF DEFORMITY IN CLUB-FOOT

Deformity	Number	Per cent. of Correction
Varus	21	42.1
Cavus	21	68.8
Equinus	20	77.7

upon an average of eight years later. Since these patients usually become bedridden as a result of paralysis and deformity in the fourth decade of life, and since the authors' examination was carried out at an average age of 22.3 years, we have no indication of the effect of the progress of the disease on the foot following arthrodesis.

The only failures of fusion occurred at the talonavicular joint. Pseudarthrosis appeared at this joint in seven instances (31 per cent.). Insufficient immobilization and poor contact of raw bone surfaces at operation were apparent. In three of the seven cases of pseudarthrosis, avascular necrosis of the navicular was an associated finding. These studies warrant no conclusion as to the relation of the high incidence of non-union and the loss of bone conduction as the disease progresses (Table VIII).

Equinus and cavus deformities showed a lower percentage of correction than in any of the other groups studied (Table IX). Avascular necrosis followed seven of twenty-two operations (32 per cent.) and was far more common than in any other group. Friedreich's ataxia *per se* is not a contra-indication to foot stabilization, although the percentage of failures is higher than in other groups (Figs. 18-A and 18-B).

FIG. 20-A

FIG. 20-B

Fig. 20-A: Pseudarthrosis of talonavicular joint and ankle joint.
Fig. 20-B: Subtalar and talonavicular pseudarthrosis of left foot.

FIG. 20-C

Talonavicular, calcaneocuboid, and probably subtalar pseudarthrosis.

TABLE XII

RESULTS OF FOOT STABILIZATION IN PAINFUL FLAT-FOOT

Type of Procedure	No. of Procedures	Average Length of Follow-up (*Years*)	Evaluation		
			Good	Fair	Failure
Subtalar arthrodesis	2	8.3	0	2	0
Hoke stabilization	5	5.4	5	0	0
Triple arthrodesis	6	1.5	5	1	0
Total	13	4.0	10 (100%)	3	0

Congenital Club-Foot (Table X)

Surgery was performed at an earlier age than in any other group in this series,—at an average of 11.5 years. Each patient had had some type of previous treatment, most often stretching, manipulation, and plaster.

The low percentage of correction of varus deformity (Table XI) can be attributed largely to the use of mid-tarsal wedge osteotomy, which cannot correct varus of the hind part of the foot.

Painful Flat-Foot

Painful congenital weakness of the feet accounted for seven cases, calcaneonavicular coalition for five, and congenital talocalcaneal fusion for one. Pain, rather than paralysis or deformity, was the sole indication for operation in these patients. In every instance but one, this symptom was completely relieved, and it was significantly improved in the remaining case (Figs. 19-A and 19-B). No procedure was graded as a failure (Table XII).

FIG. 21

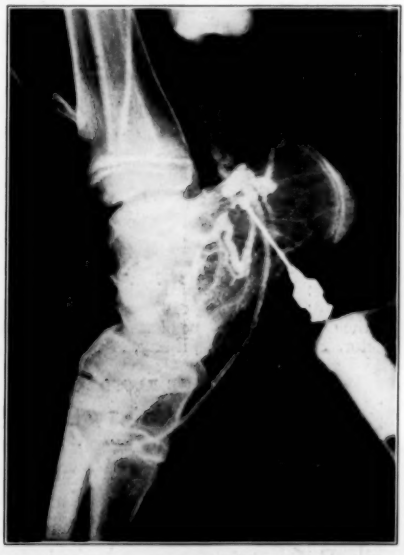

FIG. 22

Fig. 21: Avascular necrosis of talus. Subsequent ankle fusion was required.
Fig. 22: Arteriovenous fistula following stabilization.

FIG. 23

FIG. 24

Fig. 23: Rocker-bottom deformity following triple arthrodesis in a severe cerebral-spastic deformity.
Fig. 24: Loose body in ankle joint.

FIG. 25

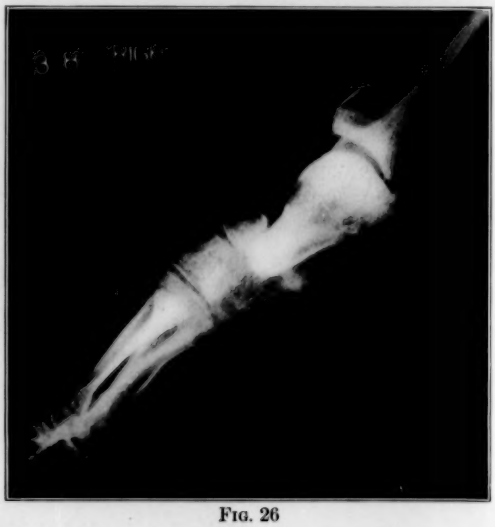

FIG. 26

Fig. 25: Draining painful callosity.
Fig. 26: Wide gaping of the ankle joint anteriorly, following foot stabilization. Neither the etiology of the condition, the type of stabilization, or any postoperative deformity appeared to be the causative factor. The significance, if any, of this asymptomatic finding is as yet undetermined.

AGE FACTOR AS AN INFLUENCE IN STABILIZATION

Arthrodesis was done eighty times before the age of twelve. Table XIII shows that, in the patients up to the age of nine, 47 per cent. of the stabilizations were failures, while in the group from nine to twelve years, only 9 per cent. were failures. The over-all average for the total of 289 primary procedures was 17 per cent. of failures. In every failure in both younger-age groups, undercorrection of the deformity was found responsible. The principal problems in the younger-age groups are the small size of the structures, the difficulty in adequately holding them in plaster in the proper position, and inability to determine muscle power accurately, as a result of which a deforming influence may persist.

TABLE XIII

AGE FACTOR IN STABILIZATION

Age Group	Results			No. of Pseudarthroses	Foot-Length Discrepancy
	Good	Fair	Failure		
8 years and under (17 cases)	5 (29%)	4 (24%)	8 (47%)	10 (12.5%)	Minus 11/16 inch
9 to 11 years, inclusive (63 cases)	39 (62%)	18 (29%)	6 (9%)		
All ages (Average 16.1 years) (269 cases)	140 (52%)	83 (30.9%)	46 (17.1%)	52 (19%)	Minus 11/16 inch

The foot-length discrepancy averaged the same in the younger group as in the whole series, so growth apparently was not interfered with.

COMPLICATIONS AND SEQUELAE

Untoward problems secondary to foot stabilization are seldom mentioned in the literature. On the whole, the complications have been few and there has been no loss of life or limb in the present series. Following the Kocher incision, wounds did not always heal by first intention, but this delay presented no problem. Hemorrhage and infection have both been present in the series. One patient required a second operative procedure to control bleeding, and another had sufficient infection to demand a secondary procedure for the removal of bone. Varying degrees of skin sloughing occurred, but in only one instance was a flap graft required. This was in a poliomyelitic patient who had had a one-stage panarthrodesis and who probably had impaired circulation at the outset.

Other common complications were injury to the peripheral nerves and pseudarthrosis. Of the former, injury to the sural nerve was more frequent, but damage to the branches of the superficial peroneal nerve can also be a source of difficulty. These nerves may be injured or caught by a suture or in scar tissue, resulting in anaesthesia, paraesthesia, neuromata, and exquisitely tender areas on the foot. In a few patients, exploration of the nerve was required for this complaint. Pseudarthrosis, discussed elsewhere, may be accompanied by pain and swelling (Figs. 20-A, 20-B, and 20-C).

Avascular necrosis has been observed at one time or another in each of the cuneiforms, the cuboid, talus (Fig. 21), and navicular. It has not always resulted in non-union, however. It was more common following operations performed in cases of Friedreich's ataxia. In three patients showing marked chronic oedema of the extremity, which dated from the time of surgery (all were examined several years after operation), the condition was considered to be the result of chronic phlebitis. In one of these patients, the difficulty has subsequently been relieved by lumbar parasympathetic blocks. Fatigue fracture of the tibia at the site of the sliding graft occurred in 15 per cent. of the cases of panarthrodesis. Arteriovenous fistula (Fig. 22) of the posterior tibial artery occurred in one patient, no doubt as a result of operative injury of the vessels, and ligation was required. Two patients complained of severe calf atrophy and loss of strength in the extremity following operation. In one of these patients the extremity had been measured before operation, and his contention was borne out. Rocker-bottom deformity (Fig. 23) was caused several times when forced dorsiflexion was employed to overcome fixed equinus, instead of removing sufficient bone. Osteochondritis dissecans with a loose body in the ankle joint was seen a number of times (Fig. 24). Painful callosities (Fig. 25) may result in severe discomfort to the patient.

Finally, gaping of the ankle joint anteriorly (Fig. 26) has been recognized in numerous postoperative roentgenograms.

REVISION PROCEDURES

Thirty-six operations were done to correct failures of previous primary stabilizing procedures. The etiology of the original deformity appeared to be unrelated to the necessity for revision. In this group the primary operation was performed at an average of 10.5 years of age, with the revision at an average of 6.3 years later.

The indications for revision were as follows:

Recurrence of deformity	28
Pseudarthrosis	18 (8 were painful)
Overcorrection of deformity	4
Different deformity	2
Painful callosity	1

Some patients had more than one indication for revision.

The revision consisted of repair of the pseudarthrosis, removal of adequate bone wedges to correct deformities, and soft-tissue procedures as indicated. Postoperative management, immobilization, complications, and sequelae were no different than in the primary procedures.

These patients were re-examined on an average of 5.5 years after revision. Nine complained of some degree of pain, seven of fatigue, and two of instability. Four pseudarthroses were found at the talonavicular joint. Correction of the deformity was good in fifteen cases, fair in sixteen, and poor in five. Varus and drop-foot were again the deformities most difficult to correct.

Seventeen revisions were rated as good, ten as fair, and nine as failures. The 75 per cent. of satisfactory results obtained justify revision after failure of a previous stabilizing procedure.

A STUDY OF THE FAILURES

In many cases more than one factor was responsible for the failure, but in each instance the authors have tried to assign the failure to its outstanding cause for the sake of clarity (Table XIV). Individual cases have been discussed in detail in the section dealing with primary bone procedures.

In thirty-nine cases the failure was due to residual deformity, and in two-thirds of this number the cause was undercorrection of the deformity by the surgeon at the time of operation. With two exceptions—mid-tarsal wedge osteotomy and subtalar arthrodesis—the type of procedure chosen to abolish the deformity seemed to have no bearing on the end result.

Recurrence of deformity was the result of more than one factor in the majority of instances. In the present series, these factors were, in order of frequency, failure to immobilize the foot until bony union was firm, failure to align the foot with the ankle mortise, loss of position in plaster or at the time the plaster was changed, poor bone contact with resultant pseudarthrosis, muscle imbalance, and, finally, the operation being carried out at too early an age. The case with overcorrection was a result of pseudarthrosis and muscle imbalance.

Pain of a severe nature could be explained in all of the cases of failure. In eight cases it was at the site of a pseudarthrosis and in the other two at the site of painful callosities. Minor degrees of undercorrection may result in callosities which are so painful that the outcome must be rated as a failure. We have no ready explanation as to why some of the pseudarthroses were painful while others were not. Of fifty-six failures of fusion in the entire series, in only ten was there sufficient pain for the procedure to be considered a failure. Eight of these cases have been included under the heading "pain at site of pseudarthrosis"

TABLE XIV

PRINCIPAL CAUSE OF FAILURE IN FIFTY-FIVE PATIENTS

Cause of Failure	No. of Patients
Residual Deformity	
Undercorrection	26
Insufficient bone removed from proper areas	
Recurrence	12
Inadequate period of immobilization	
Failure to align foot with ankle joint	
Loss of position in plaster or at change of plaster	
Failure of fusion	
Muscle imbalance	
Operation at too early an age	
Overcorrection	1
Muscle imbalance (at times secondary to injudicious muscle transplants)	
Pseudarthrosis	
Pain	
At site of pseudarthrosis	8
At site of callosities	2
Severe Instability	2
Pseudarthrosis of the Subtalar Joint (accompanied by severe pain in two cases)	4

in Table XIV, and the other two will be mentioned shortly. Instability was severe enough in only two cases for the result to be graded as a failure.

The authors are so certain that failure of fusion of the subtalar joint will eventually result in pain, instability, or recurrence of deformity that they believe pseudarthrosis of this joint in itself constitutes a failure. This condition developed in four patients in the present series, two of whom complained of severe pain.

Any attempt to compare the failures resulting from the different types of primary bone procedures is difficult and probably of little value. However, if we study the four operations which utilize the talonavicular, calcaneocuboid, and talocalcaneal joints as one group, we find the percentage of failures to be quite similar (Hoke stabilization 12.5 per cent., Brewster countersinking operation 20.0 per cent., Lambrinudi stabilization 14.5 per cent., and triple arthrodesis 15.7 per cent.). Undercorrection at the time of operation was usually the common denominator of this group. If we compare the percentage of failure in this group with that from subtalar arthrodesis (31.25 per cent.) and wedge osteotomy (34.8 per cent.), some explanation should be sought for the sharp rise of poor results. The shortcomings of these two procedures in overcoming deformity have already been mentioned. Neither completely mobilizes the foot so that any desired position can be obtained, nor does either of them allow rotation of the calcaneus beneath the talus so that the foot can be properly aligned with the ankle. The authors believe panarthrodesis to be the most difficult operation, from a technical standpoint, of all the bone operations studied. If it is properly done, the foot should be broken into four "pieces" instead of three. It is reasonable to assume that it is much more difficult to gain and maintain good contact, position, and alignment, plus final consolidation, in four joints than it is in three. More dexterity is required in resecting the various wedges so that all the raw bone surfaces fit together accurately; and, even if this can be accomplished, some form of internal fixation may not be amiss. The authors believe that this operation is best carried out in two stages.

CONCLUSIONS

1. An evaluation of 305 foot stabilizations, based on anatomical and functional end results, shows that stabilization was successful in 82 per cent. of the cases. Residual de-

formity or pseudarthrosis accounted for fifty-one of fifty-five failures. Over two-thirds of the residual deformities resulted from undercorrection at the time of operation. Listed in order of frequency, removal of the plaster before solid fusion had occurred, failure to align the foot with the ankle joint, pseudarthrosis, and loss of position in plaster or at the change of plaster were much more common as the causes of recurrence of deformity than muscle imbalance.

2. The results were more successful when three joints were resected. Mid-tarsal wedge osteotomy resulted in the highest percentage of failure and did not correct the foot varus.

3. Stabilization done on children up to and including eight years of age produced 47 per cent. of failures. In the group from nine to eleven years, inclusive, the results were as good as those in the series as a whole.

4. Varus and drop-foot were the most frequent deformities that remained undercorrected. Stabilization, with the exception of panarthrodesis, cannot be expected to correct drop-foot.

5. Pseudarthrosis occurred in 18 per cent. of the cases. Approximately one-fifth of the patients had such severe pain that a rating of failure was mandatory. In the fifty-six pseudarthroses, 89 per cent. were at the talonavicular joint.

6. Three-quarters of the revision procedures produced satisfactory results, indicating that repeated intervention was justified.

7. Measurement of the foot which had been operated upon disclosed that it averaged eleven-sixteenths of an inch shorter than the foot which had not been operated upon. The same foot-length discrepancy was present in eighty patients who had been operated upon before the age of twelve years.

8. These operations have been employed for many conditions, but by far the most common, in order of frequency, were poliomyelitis, spastic paralysis, Friedreich's ataxia, congenital club-foot, and painful flat-foot.

REFERENCES

1. Brewster, A. H.: Countersinking the Astragalus in Paralytic Feet. New England J. Med., **209**: 71–74, 1933.
2. Fitzgerald, F. P., and Seddon, H. J.: Lambrinudi's Operation for Drop-Foot. British J. Surg., **25**: 283–292, 1937.
3. Guildal, Paul, and Sodemann, Thor: Results of 256 Triarticular Arthrodeses of the Foot in Sequelae of Infantile Paralysis. Acta Orthop. Scandinavica, **1**: 199–230, 1930–1931.
4. Hallgrímsson, Snorri: Studies on Reconstructive and Stabilizing Operations on the Skeleton of the Foot. With Special Reference to Subastragalar Arthrodesis in the Treatment of Foot Deformities Following Infantile Paralysis. Acta Chir. Scandinavica, **88**: Supplementum 78, 1943.
5. Hamsa, W. R.: Panastragaloid Arthrodesis. A Study of End Results in Eighty-five Cases. J. Bone and Joint Surg., **18**: 732–736, July 1936.
6. Hart, V. L.: Lambrinudi Operation for Drop-Foot. J. Bone and Joint Surg., **22**: 937–941, Oct. 1940.
7. Hoke, Michael: An Operation for Stabilizing Paralytic Feet. J. Orthop. Surg., **3**: 494–505, Oct. 1921.
8. Lambrinudi, C.: New Operation on Drop-Foot. British J. Surg., **15**: 193–200, 1927.
9. Lambrinudi, C.: A Method of Correcting Equinus and Calcaneous Deformities at the Sub-Astragaloid Joint. Proc. Royal Soc. Med., **26**: (Sect. Orthop.): 788–791, 1933.
10. Liebolt, F. L.: Pantalar Arthrodesis in Poliomyelitis. Surgery, **6**: 31–34, 1939.
11. Ryerson, E. W.: Arthrodesing Operations on the Feet. J. Bone and Joint Surg., **5**: 453–471, July 1923.
12. Steindler, Arthur: The Treatment of the Flail Ankle; Panastragaloid Arthrodesis. J. Bone and Joint Surg., **5**: 284–293, Apr. 1923.
13. Steindler, Arthur: A Textbook of Operative Orthopedics, p. 250. New York, D. Appleton and Co., 1925.
14. Stephens, Richmond: An Operation for the Correction of Pronated Feet. J. Bone and Joint Surg., **17**: 424–426, Apr. 1935.
15. Thompson, T. C.: Astragalectomy and the Treatment of Calcaneovalgus. J. Bone and Joint Surg., **21**: 627–647, July 1939.
16. Wagner, L. C.: Modified Bone Block (Campbell) of Ankle for Paralytic Drop-Foot. With Report of Twenty-seven Cases. J. Bone and Joint Surg., **13**: 142–148, Jan. 1931.

PROGRESSIVE MUSCULAR ATROPHY OF THE PERONEAL TYPE (CHARCOT-MARIE-TOOTH DISEASE)

Orthopaedic Management and End-Result Study *

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Ordinarily the outstandingly disabling feature of the peroneal type of progressive muscular atrophy is the foot deformity, second to muscle weakness and imbalance. Since the independent reports of Charcot and Marie and of Tooth, in 1886, interest in this familial disease has centered largely in the genetic and diagnostic aspects, and yet it is to reconstructive surgery that those afflicted look for help.

References in the literature to surgical management are rare and inexact, and most writers have covered the phase of "treatment" with an assertion that the disease is progressive and eventually disabling and that treatment thus consists in the application of braces and supports. The authors, who disagree with these observations, desire to present evidence that properly designed surgical treatment can correct, prevent, or materially reduce disability in the majority of cases. Of the eighty cases reported here,† all but five, none of them treated surgically, have either been reviewed or the patients have been interviewed in the last year. Forty-five cases were treated surgically. Twenty-five of these patients have been followed for over five years,—fifteen for ten years or longer. The longest period of observation was nineteen years.

HISTORICAL DATA

Charcot and Marie, in their original paper, described the condition as progressive muscular atrophy, beginning in the feet and legs and affecting the hands and forearms only after a lapse of several years. Their opinion was based upon the personal observation of five patients; in addition, from a review of the literature, they had found a total of seventeen cases, in four different families. In his Cambridge thesis of the same year, Tooth described the condition independently on the evidence of five personal observations. He emphasized the frequency with which the peroneal muscles are first attacked and proposed the title: "The Peroneal Type of Progressive Muscular Atrophy". Herringham projected the genetic aspect in 1888, reporting a study of four generations in one family in which only males were affected.

Since that time numerous writers [2, 8–11, 13, 15, 18, 19, 23, 25, 26, 28, 30] have described one or more new features. In 1926, Roussy and Lévy presented what they considered to be a new entity in that they found cases of familial claw-foot with absence of tendon jerks. Symonds and Shaw reviewed the literature in an effort to show that this entity of Roussy and Lévy was in reality a *forme fruste* of Charcot-Marie-Tooth disease.

Considerable attention has been focused upon channels of transmission. Allan, working on clinical material supplied in part from the case histories used in this report, con-

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 25, 1949.

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† Twenty-three of these cases have been treated at the North Carolina Orthopaedic Hospital in the past twenty years, and the authors are indebted to its Chief, Dr. William M. Roberts, for use of this material. The remaining cases are taken from the files of the Miller Orthopaedic Clinic.

cluded, in 1938, that the disease was due to unit traits conditioned by a single defective gene; based upon a wide survey, he showed a triple pattern in inheritance: dominant, sex-linked recessive, and simple recessive. Allan concluded that "the pattern of inheritance determines the age of onset and the clinical severity of various morbid unitary traits". If this is true, and it has not been denied, a study of inheritance patterns in each case will be of considerable prognostic value.

Ford, in 1937, had described a similar triple pattern of inheritance, but found only one instance—that reported by Herringham—in which the condition was sex-linked recessive. Crank and Reider reported an additional family in which the trait was sex-linked. Schwartz, in 1944, reviewed another case in a family, first reported by Macklin and Bowman in 1926. On the basis of this one family, involving five generations and 101 individuals, of whom twenty-one were affected by nerve atrophy, he concluded that the condition was not sex-linked and recessive, but was due to a unit character on a fifty-fifty ratio, in which half the offspring of the affected individuals would be expected to be affected.

The authors' observations tend to confirm Allan's findings of a triple pattern, possessing prognostic value.

Because of the inevitable progress of this disease to a crippling condition, surgical treatment had been neglected until Miller, in 1924, reported four cases treated by stabilization and tendon transfers. Miller, taking friendly issue with the then current textbook of Jones and Lovett, stated: "In our experience the feet reach their maximum improvement, both in appearance and function, by having subastragaloid arthrodeses, foot stabilizations, in addition to any necessary plastic tenotomies".

SIGNS AND SYMPTOMS

The original description of the disease by Charcot and Marie and by Tooth has been little improved upon. As the disease advances in the lower extremities there is, in addition to atrophy of the peroneal muscles, involvement of the tibialis anterior, the extensor digitorum longus, and the gastrocnemius. Because of the pattern of muscle wasting, the early foot deformity is usually pes equinovarus with rapidly developing pes cavus. There is often clawing of the toes. The upper extremity demonstrates first a loss of power of the intrinsic hand muscles and symmetrical atrophy of the forearm muscle groups. The characteristic attitude of the hands is one of mild clawing, a "monkey fist". Symonds and Shaw quoted Charcot and Marie as stating that there is relative integrity of the proximal groups of muscles and absolute integrity of the trunk, shoulders, and face. Because of this distal wasting and proximal integrity, the characteristic "fat bottle" or "inverted champagne bottle" appearance of the limbs develops.

The progress is steady after onset, which varies apparently with the type of inheritance pattern[1]. In the present series the earliest age at which onset was noted was under one year. Symptoms were recorded in twenty-one patients under five years of age, twelve males and nine females. In the rest the onset was more insidious and the exact year cannot be stated, but in only two patients, one forty and one fifty-seven years of age, were the symptoms not evident before the age of twenty. The time elapsing between the first symptoms of the affliction and the application for relief varied greatly. As this is influenced by many factors, principally economic and educational, it is impossible to draw a worthwhile conclusion as to the rapidity of crippling after the initial onset. The chief complaint is usually difficulty in walking and in obtaining shoes.

The symmetrical and peripheral distribution of the atrophy at once suggests the diagnosis. In the wasting muscles fibrillar twitching may be present, and in the advanced segments there is noticeable absence of muscle contracture. As a rule, gross sensations are normal, but in some instances definite areas of hypoaesthesia and loss of vibratory sense have been found. Although absolute anaesthesia in small areas has been reported, the picture is primarily that of motor paralysis.

The question of tendon reflexes has aroused considerable debate through the years [24, 29]. Charcot and Marie reported that the knee jerks were absent in two cases, present but diminished in two, and in the fifth case feebly present in one extremity and absent in the other. They stated that the absence of the jerks was observed even before the muscles upon which they depend had undergone marked atrophy or noticeable diminution in their function. Ankle jerks were not mentioned. In the upper extremities the triceps reflex was maintained in three of the cases, absent in one, and "doubtfully" present in the fifth. In Tooth's cases the knee jerks were present in three, variable in one, and altogether absent in the other. He did not mention the ankle or upper-extremity reflexes.

In the present series the earliest observations are not always recorded, but follow-up examinations have convinced the authors that, as the motor power in the muscle wanes, there will be a loss of tendon reflex, as would be expected. We do not believe this loss is quantitative, and it is of little diagnostic or prognostic value. The reaction of degeneration is reported to occur rather early, as would be expected in a primary neurogenic atrophy.

PATHOLOGICAL FINDINGS

The primary lesion is assumed by Bowden and Gutmann to be a degeneration of the anterior horn cells with peripheral degeneration of the motor nerves. However, in contradiction to this statement, the authors have been unable to find positive evidence in the literature of histopathological changes in the spinal cord, and they have not had the opportunity to examine necropsy material. In a differential study of the primary and secondary myopathies, with biopsies, Bowden and Gutmann stated: ". . . the nerve trunks are empty or contain both normal fibers and empty Schwann tubes. They may, however, occasionally contain regenerated nerve fibers. This may indicate that some of the anterior horn cells have not undergone irreversible changes but are capable of sending out new axons." The histological examination of a branch of the common peroneal nerve in one of the authors' cases does not show abnormalities. Special stains for axon cylinders (Bodian's technique) and stains for myelin sheaths fail to reveal evidence of pathological changes. A rather marked mucinous degeneration of the endoneurium is found. Although this is very conspicuous, it is not considered to be of specific significance, since similar changes have been frequently observed by the authors in sections of peripheral nerves.

Bowden and Gutmann found little atrophic change in the muscle fibers, but some increase in connective and fat tissue, in contradistinction to the almost total disappearance of normal muscle pattern in primary myopathies.

Histological examination in one of the authors' cases (Figs. 1-A and 1-B) shows that the muscle bundles for the most part are intact. There is a mild increase of fat tissue between the muscle bundles, but the fibers themselves are not separated by fat tissue. In some areas the caliber of muscle fibers varies from three to thirty-five micra. The narrower muscle fibers show a great increase of nuclei in the sarcolemma, indicating an attempt at regeneration. Actual degenerative changes in the sarcoplasm are not encountered.

The etiology of the destruction and the mechanism of the selective pattern (centripetal atrophy) is not clear.

ROENTGENOGRAPHIC FINDINGS

Currie noted in a case of Charcot-Marie-Tooth disease (in a patient aged nine) definite clinical and roentgenographic evidence of atrophy of the tibia and fibula, the lower end of the femur, and, to a lesser extent, of the radius and ulna. The bone atrophy corresponded in distribution to the muscular atrophy.

In ten cases of the present series, no significant bone atrophy was noted by roentgenogram, nor was the tibia or fibula smaller as compared with these bones in children of the same age group. At the time of surgery, the bones certainly appeared as dense as those in other feet.

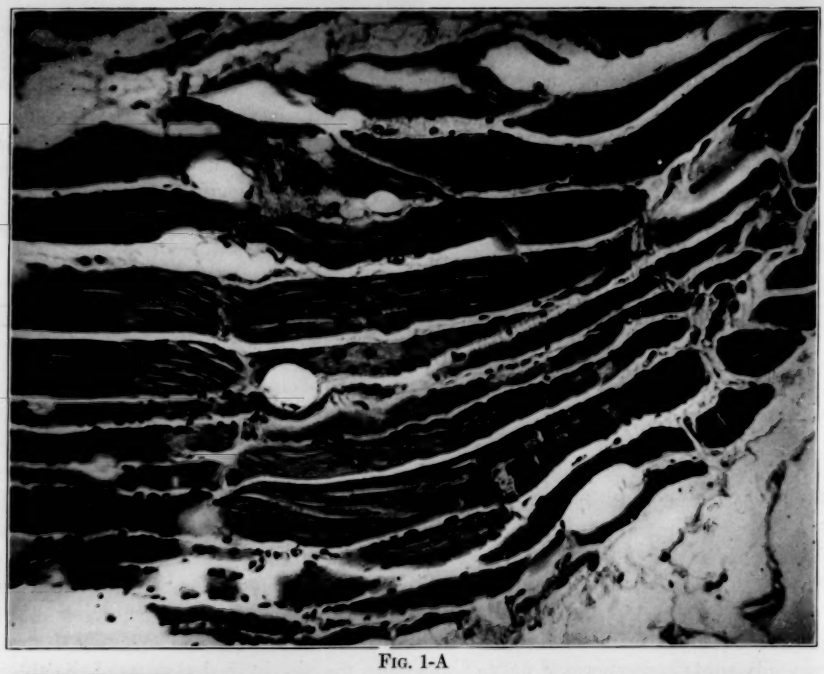

FIG. 1-A

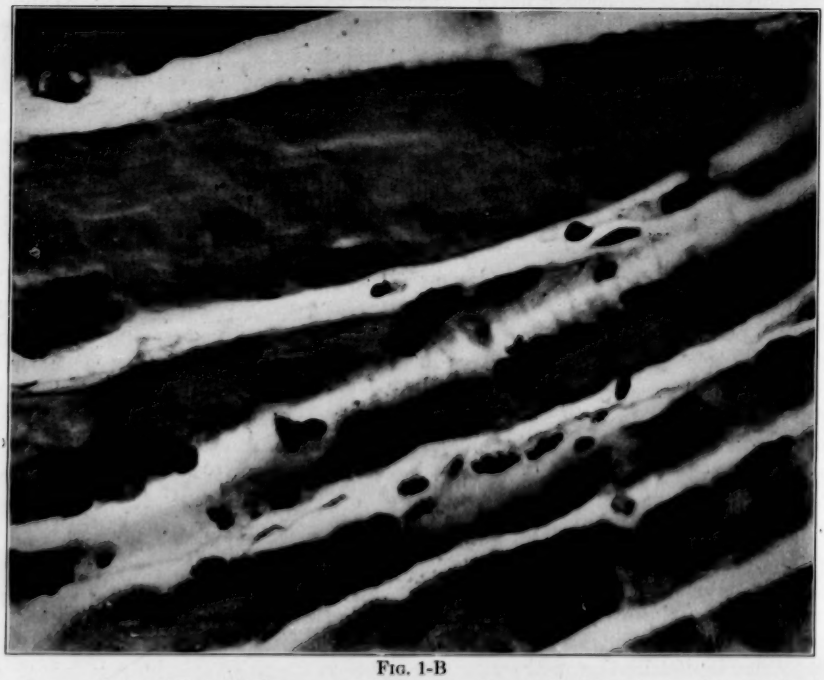

FIG. 1-B

Low-power (× 275) and high-power (× 1000) photomicrographs. There is atrophy of striated muscle fibers. Under low-power magnification (Fig. 1-A) one can detect a marked variation in the caliber of the fibers, which vary from 3 to 35 micra in diameter. The narrow muscle fibers are atrophic, but show a greatly increased number of nuclei in the sarcolemma, evidence of an attempt at regeneration. The high-power magnification (Fig. 1-B) shows more clearly the variation in the caliber of muscle fibers and the increased nuclei.

HEREDITARY FACTORS

In his work on inheritance factors, Allan reached conclusions of such value as to be worthy of repetition:

"The afflicted members of the family in which peroneal atrophy is dominant notice beginning disability about the age of 30 but are able to plough their fields and pick cotton past the age of 70. The atrophy is moderate, or there may be only pes cavus.

"When peroneal atrophy is sex-linked recessive, the boys notice atrophy about the middle of the second decade and by the time they are 25 are too disabled for such occupations as watchmaking and bookkeeping, are on crutches or are bedridden.

"When it is a simple recessive trait, peroneal atrophy begins before the age of 8 years, and in the second decade its victims become hopeless cripples.

"The explanation seems to be this: Those in whom the trait is dominant, who are all heterozygous in case of these uncommon diseases, receive one defective gene from the affected parent and one normal gene from the unaffected parent; such a pair of genes, half good and half bad, causes half blindness or half crippling with late onset.

"The boys with a sex-linked recessive trait, of course, receive one defective gene from their mother, without any corresponding normal gene on the chromosome they receive from their father, and the trait is correspondingly more severe, with an earlier onset.

"When the trait is simple recessive, each parent supplying a defective gene, there is nothing to ameliorate the intensity of the disease, and it is very severe, with an early onset. This behavior of the genes explains the relation of hereditary pattern to clinical severity and age of onset."

In the present series the inheritance, unfortunately, has not been fully traced, and in many of the earlier cases the family histories were meager and furnished inconclusive evidence. Of the eighty cases reported, a positive family history was recorded in thirty-six. Ten cases were treated in which one or more siblings were involved and are included in this series. In all, there were thirty-three females and forty-seven males. Operation was performed on twenty-five females and twenty males. In two cases included in the series, operation was performed elsewhere.

At the North Carolina Orthopaedic Hospital Clinic, among approximately 3,000 negroes examined for orthopaedic conditions in the period covered by this review, no positive instance of progressive muscular atrophy of the peroneal type has been seen. No reference to such occurrence in negroes has been found in the literature. Mirvish, reporting one family from South Africa, failed to mention the race, but one would infer from the names that the family was of Dutch descent.

The authors conclude, at least on the evidence thus far presented, that the colored race is exempt from this hereditary affliction. This factor is difficult to evaluate, since consanguinity in the negro must be at least as common as in the closely bred Anglo-Saxons of Scotch-Irish extraction comprising the subjects in the present series. Even if the negro race proves to be free of this condition, the multiplicity of reports from all over the world would indicate that no other race is exempt. This knowledge of hereditary factors is useful, both as a prognostic aid and as a yardstick of results.

INVOLVEMENT

All the patients seen had bilateral pes equinovarus and pes cavus. In those at the North Carolina Orthopaedic Hospital, all of whom were children, a contracted triceps surae with some equinus was present in nearly every case, but in only three was clawing of the digits distressing and in none of these cases have claw-toe procedures *per se* been performed. In contrast, in older groups seen at the Miller Orthopaedic Clinic, surgery for the correction of claw-toe has been done in six cases (twelve feet), and in at least ten more cases such procedures have been recommended. In this series the hands were involved in

thirty-six cases when the patients were first seen. Deformities eventually appeared in the hands of all, but not to the same disabling extent as in the feet.

In these cases no corrective surgery has been carried out for the upper extremities. The authors' observations have been largely confined to the lower extremities, and further discussion in this paper will be limited to the lower extremity.

Muscle evaluation regularly shows a pattern of loss corresponding to the extent of the deformity. The peroneal muscles are weak to absent; the anterior tibial muscles are usually fair; the posterior tibial muscle and the calf groups are present and good. There is general weakness in all foot muscles. Although the clawing factor is difficult to evaluate, it seems to originate in weak intrinsic muscles and weakening flexors, while the toe extensors remain strong and overactive. In one case there was complete foot-drop from dorsiflexor insufficiency.

FIG. 2-A

FIG. 2-B

Close-up views of feet, before and after stabilization and tendon transplantation.

In only one case was scoliosis noted and only one patient is bedfast, at the age of seventy-two. One man, aged fifty-nine, is still doing all his own farm work. Two patients, aged forty-nine and thirty-three, who were not operated upon, are crippled but not bedfast. One patient, aged twenty, is crippled as a result of resection of all metatarsal heads, performed elsewhere. In all the other cases followed the patients are ambulatory.

The most advanced involvement occurred in two sisters, U.B. and E.B. (Figs. 2-A, 2-B, 3-A, 3-B, 3-C, and 3-D), who were operated upon in 1928 at the ages of fourteen and sixteen. Genealogy charts showed that these patients represented a simple recessive inheritance. The younger sister, E.B., was seen in 1947 (Figs. 3-C and 3-D). She was then thirty-three years of age and was single. Although the disease had advanced, the feet had not become deformed. She was brace-free, and was able to get around unassisted and to do light housework. The other sister, aged sixteen at the time of her foot-stabilization procedure, has not been seen since she was nineteen. When she was thirty-five, her sister and mother stated that she had been married for eight years. She had no children. She was stronger than the other sister and did all her own housework. According to Allan's deductions, if the disease is inherited as a simple recessive trait, these patients, if left alone, would have been hopeless cripples in the second decade.

SURGICAL PROCEDURE

The authors' operation has consisted of foot stabilization, preceded by plantar fasciotomy or lengthening of the tendo achillis, as indicated. Section (or transplantation forward) of the posterior tibial tendon, either alone or in combination with transplantation of the anterior tibial tendon laterally to the central tarsus, should be done. This operation has been performed, with slight variations to meet specific indications, on eighty-nine feet in forty-five patients. Most of the stabilizations were carried out on patients between the ages of seven and seventeen. Only twelve of the forty-five patients operated upon were seventeen years of age or older. In the young children it has been the custom

to await suitable bone density. The youngest patient upon whom stabilization was performed was seven years of age; the oldest, fifty-six years.

The basic part of the procedure is, we believe, the bone surgery. Miller originally brought to this work the ideas of Hoke, and the technique is often referred to as the Hoke stabilization, although G. G. Davis deserves priority in publishing the general concept of the procedure.

Briefly, after subcutaneous plantar fasciotomy and lengthening of the Achilles tendon, the tarsus is opened by an incision over the sinus tarsi. The sinus is cleared of soft tissue and the posterior subtalar joint is exposed. From this area the superior (talar) and the inferior (calcaneal) articular

FIG. 3-A

FIG. 3-B

Close-up views of feet, before and one year after reconstructive surgery.

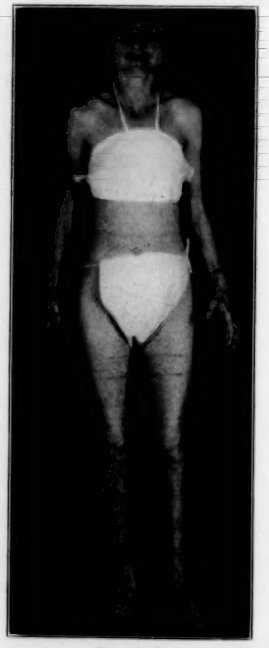

FIG. 3-C

FIG. 3-D

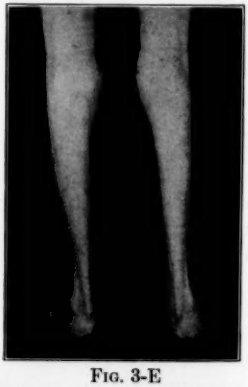

FIG. 3-E

Fig. 3-C: Photograph of E. B., aged thirty-three, nineteen years after foot surgery. (Note condition of the hands.)

Fig. 3-D and Fig. 3-E: Close-up views of feet, sixteen years after operation.

cartilage is removed. If the foot is in the varus position, slightly more subjacent bone is removed on the lateral than on the medial aspect, a shallow wedge being formed with the base laterally. The talar head is freed subcapsularly and subperiosteally by means of the Hatt spoon, and the head and neck are removed and preserved. When moderate equinus is present, the neck may be severed obliquely backward and in the plantar direction, in the sagittal plane; when the foot is repositioned, the fore part of the foot will be "brought up" to the hind portion, the principle of Lambrinudi being utilized. The exposed articulating surface of the navicular is denuded and then the calcaneocuboid joint is

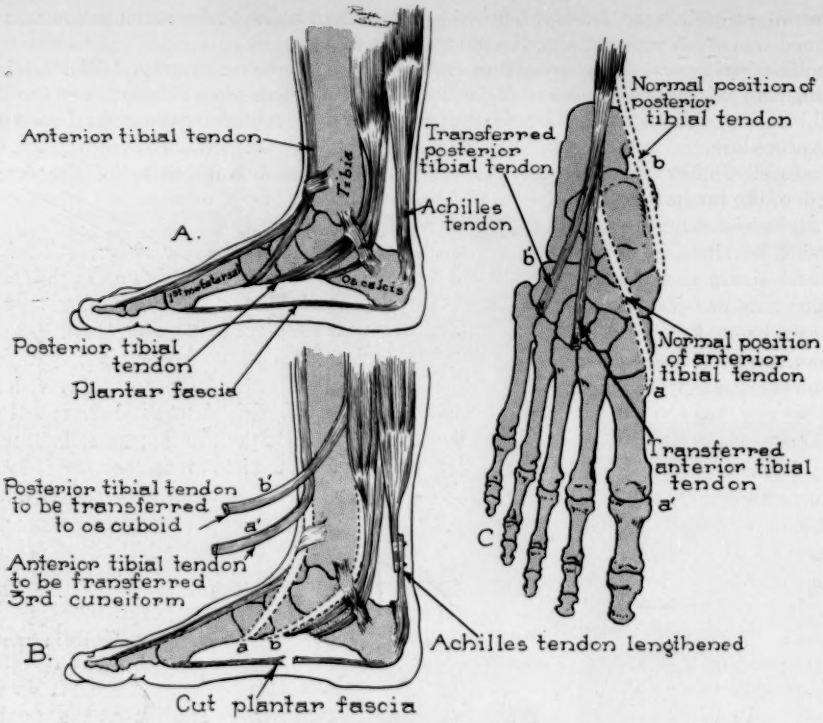

FIG. 4

Diagrammatic representation of the tendon and soft-tissue procedures used.

excised; in the varus foot the base of the wedge, although narrow, is to the lateral aspect. After this dissection and excision, the soft tissues should be so freely movable as to allow reposition of the bones for realignment of the foot. The head of the talus is denuded of cartilage and the bone is replaced as necessary to close the gaps. With the foot in the varus position, the replacement is usually to the medial side. In the majority of these feet, lowering of the arch is required, and this can be effected by shortening the neck of the talus.

In G.P., aged fourteen, a posterior bone-block operation at the ankle was performed for severe bilateral foot-drop. This is the only instance in which such a procedure was required for this type of paralytic foot; since the posterior tibial tendon has been transplanted forward, there has been no need for a bone block.

Formerly the authors transplanted the anterior tibial tendon to the mid-tarsus, countersinking the tendon by drilling a Steinmann pin (a medium-sized pin with an eye) through the tarsus and out through the sole, and fixing the tendon in bone with a "pull-out" suture tied over a rolled-up sponge; then the posterior tibial tendon was sectioned. Lately, the anterior tibial tendon has been left in place and the posterior tibial tendon has been transplanted through the interosseous space either into the cuboid or the middle cuneiform, depending upon the strength of the anterior tibial tendon (Fig. 4).

The necessary tendon transfers having been accomplished, the foot is molded into a padded toe-to-groin plaster cast, but weight-bearing is not permitted. At the end of three or four weeks the cast is removed and the foot is inspected. If indicated, the pliable union is remolded or refined, with the patient under anaesthesia, to improve the appearance of the foot, and a walking cast is applied for an additional four to six

weeks. In bilateral cases the second foot is usually operated upon at the time the first foot is molded, rather than subjecting the patient to double foot surgery at one operation.

EVALUATION OF END RESULTS

In evaluating the end results after operation, twenty-one cases have been classified as excellent, twelve as good, and twelve as poor. The latter group includes those whose results might be considered "fair", for whom additional treatment may be provided.

Satisfactory Results

In the group classified as "excellent", the feet have maintained their function and appearance, and the patients are ambulatory and brace-free.

Such a case is illustrated in Figures 5-A and 5-B. This boy, P.C., was eight and one-half years of age when he was operated upon in 1931. Although he walks with a rather stiff gait and has considerable muscle weakness, he is now able to ride a bicycle daily and to earn his living as a grocer's deliveryman.

Another case classified as "excellent" presented a milder type of involvement, mainly cavus which had progressed gradually for about four years. This patient was operated upon at the age of sixteen. Eleven years afterward her lower extremities had less than the usual

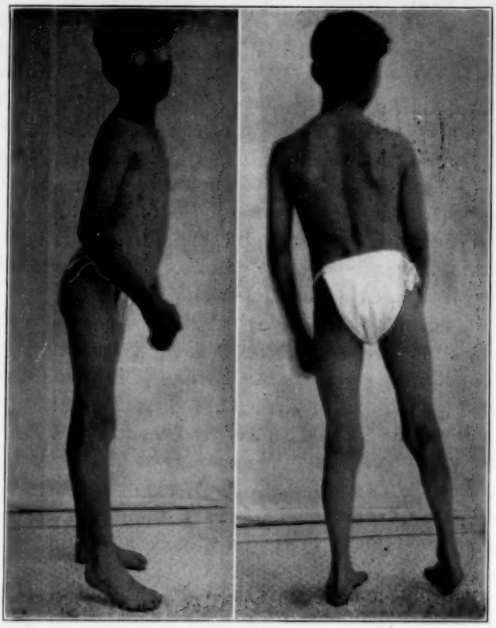

FIG. 5-A

P. C., aged eight and one-half years, before operation.

FIG. 5-B

Views of patient's feet, sixteen years later.

endurance, but she was able to care for her home and two children. Neither of the children shows evidence of the affliction.

In another such case, J.O. was operated upon in 1946, at the age of seventeen. He was followed for only two years. His first-year and second-year results can be classified as excellent, as far as the feet are concerned. He is a farm boy and has had to give up more and more of the heavier work. He does not wear a brace.

H.O., a sister of J.O., was followed for three years after surgery on her foot. The result is only "good". Clawing of the toes is troublesome, but she is able to work daily as a typist.

In the cases classified as good the patients are ambulatory, but the feet, although maintaining a weight-bearing position, are not prepossessing in appearance. Clawing of the toes has developed in most cases, and pads and bars must be used. In such a case, a man of twenty-six years, the patient was interviewed in 1947, ten years after operation. In spite of their poor appearance, the feet had good function. He earned his living as a taxi driver, but was unable to stand for a prolonged time or to walk a long distance.

In seven of the twelve good results the anterior tibial tendon was *not* transferred to the central tarsus; all of the patients have claw-toe. The relative and continued overaction of the toe extensors acting as dorsiflexors may be suspected as the mechanism of the clawing. Even in the cases classified as poor, some benefit has resulted. None of the patients wear braces. Only one who has undergone surgery is a complete cripple. That patient, not included in the forty-five, had resection of the metatarsal heads performed elsewhere.

A tendency to recurrence of the varus was found in six cases in which, for one reason or another (usually the operator's judgment), the deforming influence of the posterior tibial tendon was not removed. In Case 23, a fifteen-year-old boy, the anterior tibial tendon was not transplanted centrally on the left foot, but this was done on the right foot. In one year varus deformity had appeared on the left and secondary transfer of the anterior tibial tendon was accomplished. By August 1948, one year after this last transplantation, there had been considerable improvement, but the transplant on the left was not functioning so strongly as the two-year-old transplant on the right. Toe "cock-up" or clawing was still in evidence. The feet maintained their position.

The posterior tibial tendon has not been transferred anteriorly in any of the younger patients at the North Carolina Orthopaedic Hospital, as in only one (G.P., Case 16) was the foot-drop a factor. This is the patient, mentioned previously, who had a posterior bone block inserted.

In three cases (five feet) in the older group, the posterior tibial tendon has been transplanted forward, with the principle of always using a functioning motor. These cases seem to have had the best results so far.

Unsatisfactory Results

In twelve cases (twenty-three feet) the results were poor. One patient, mentioned previously, whose posterior tibial tendon was not sectioned or transferred primarily, was improved by having this transplant in one foot and now desires it in the second foot. One patient had anterior tarsal wedges and plantar fasciotomies with recurrence. Another underwent heel-cord lengthenings and plantar fasciotomies at eleven years of age and had bilateral stabilization at twenty-three; the result is only fair. In another case the dorsal tarsal wedges were combined with heel-cord lengthenings and there has been moderate recurrence.

At the age of twenty-two, Case 42 had bilateral heel-cord lengthening, plantar fasciotomy, and triple arthrodesis. She had no tendon transfers and the condition is recurring. In another case the result was fairly good for thirteen years with only triple arthrodesis and fasciotomy, but now, when the patient is twenty-six, tendon repair is needed.

E.H., an eight-year-old girl at the time of her operation, had the complete operative procedure on the left, but only triple arthrodesis on the right. She is now sixteen, has had a recurrence in the right foot, and is to be readmitted for correction of this deformity.

Case 54, a mill worker, aged twenty-three, had had foot surgery performed at the age of twelve. The operation included sectioning of the posterior tibial tendon. His feet function poorly. This case illustrates the authors' present procedure of transferring this tendon forward to preserve its usefulness.

In Case 16, mentioned previously, the patient had stabilization and posterior tibial section plus bilateral posterior bone block, ten years ago. The foot-drop and varus on the left recurred. This was noted as early as one year after the operation, and in this instance may be attributed to mechanical failure of the left arthroereisis. The patient is a housewife and, in spite of her disability, she carries on her duties and does light work on the farm.

Case 22, a boy, now eleven years of age, had stabilization operations only at the age of seven. The deformity is recurring sharply; the patient wears metatarsal bars and heel wedges and needs further reconstructive surgery. One must balance the foot with both bone and tendon repair or recurrences are inevitable.

SUMMARY

Of eighty cases of hereditary peroneal palsy which have been studied, forty-five patients have undergone surgery for the correction or amelioration of the crippling foot deformity. On the basis of adequate follow-up observations, the surgical procedures outlined have been shown to improve the crippling condition. In spite of progressive muscle wasting, the rehabilitation can be maintained for many years.

In twenty-three feet, the operation gave less than satisfactory results. Sixty-six feet in which adequate surgery was performed have stood the test of time in an encouraging manner.

NOTE: Appreciation is expressed to Paul Kimmelsteil, M.D., for his assistance in the pathological study, and to O. L. Miller, M.D., for valuable suggestions.

REFERENCES

1. ALLAN, WILLIAM: Relation of Hereditary Pattern to Clinical Severity as Illustrated by Peroneal Atrophy. Arch. Int. Med., **63**: 1123–1131, 1939.
2. BERNHARDT, M.: Weiterer Betrage zur Lehre von den hereditären und familiären Erkrankungen des Nervensystems. Virchow's Arch. f. Path. Anat., **133**: 259–294, 1893.
3. BOWDEN, R. E. M., and GUTMANN, E.: Observations in a Case of Muscular Dystrophy, with Reference to Diagnostic Significance. Arch. Neurol. and Psychiat., **56**: 1–19, 1946.
4. CHARCOT, J.-M., et MARIE, P.: Sur une forme particulière d'atrophie musculaire progressive souvent familiale débutant par les pieds et les jambes et atteignant plus tard les mains. Rev. de Méd., **6**: 97–138, 1886.
5. CRANK, H. H., and REIDER, NORMAN: Genetic Features in the Charcot-Marie-Tooth Type of Muscular Atrophy. Bull., Menninger Clin., **3**: 88–91, 1939.
6. CURRIE, R. A.: Case of the Charcot-Marie-Tooth Type of Muscular Atrophy, with a Note on the Condition of the Bones. Glasgow Med. J., **107**: 28–30, 1927.
7. DAVIS, G. G.: The Treatment of Hollow Foot (Pes Cavus). Am. J. Orthop. Surg., **11**: 231–242, Oct. 1913.
8. DEJERINE et ARMAND-DELILLE: Un case d'atrophie musculaire, type Charcot-Marie, suivi d'autopsie. Rev. Neurol., **11**: 1198–1201, 1903.
9. DUNCAN, G. A.: Orthopaedic Treatment of the Muscular Dystrophies and Muscular Atrophies. Virginia Med. Monthly, **64**: 40–41, 1937.
10. EISENBUD, ADOLPH, and GROSSMAN, MORRIS: Peroneal Form of Progressive Muscular Atrophy. A Clinical Report of Two Families. Arch. Neurol. and Psychiat., **18**: 766–778, 1927.
11. FORD, F. R.: Diseases of the Nervous System in Infancy, Childhood and Adolescence. Springfield, Illinois, Charles C. Thomas, 1937.
12. HERRINGHAM, W. P.: Muscular Atrophy of the Peroneal Type Affecting Many Members of a Family. Brain, **11**: 230–236, 1888.
13. HOFFMANN, J.: Ueber progressive neurotische muskelatrophie. Arch. f. Psychiat., **20**: 660–713, 1889.
14. HOKE, MICHAEL: An Operation for Stabilizing Paralytic Feet. J. Orthop. Surg., **3**: 494–505, Oct. 1921.

15. Ingham, S. D.: A Case of Transition Between Charcot-Marie-Tooth and General Form of Progressive Spinal Muscular Atrophy. Bull. Los Angeles Neurol. Soc., **3:** 136–137, 1938.
16. Jones, Robert, and Lovett, R. W.: Orthopedic Surgery. New York, William Wood and Co., 1923.
17. Lambrinudi, C.: New Operation on Drop-Foot. British J. Surg., **15:** 193–200, 1927.
18. Macklin, M. T., and Bowman, J. T.: Inheritance of Peroneal Atrophy. J. Am. Med. Assn., **86:** 613–617, 1926.
19. Milhorat, A. T.: Studies in Diseases of Muscle. XIV. Progressive Muscular Atrophy of Peroneal Type Associated with Atrophy of the Optic Nerves; Report on a Family. Arch. Neurol. and Psychiat, **50:** 279–287, 1943.
20. Miller, O. L.: Reporting Cases of an Infrequent Type of Foot Deformity. Southern Med. and Surg., **86:** 512–514, 1924.
21. Miller, O. L.: Surgical Management of Pes Calcaneus. J. Bone and Joint Surg., **18:** 169–172, Jan. 1936.
22. Mirvish, L.: Familial Incidence of Peroneal Type of Progressive Muscular Atrophy (Charcot-Marie-Tooth Disease). South African Med. Rec., **23:** 411–412, 1925.
23. Moleen, G. A.: Familial Progressive Muscular Atrophy. Arch. Neurol. and Psychiat., **27:** 645–659, 1932.
24. Roussy, Gustave, et Lévy, Gabrielle: Sept cas d'une maladie familiale particulière: Troubles de la marche, pieds bots et aréfléxie tendineuse généralisée, avec, accessoirement légère maladresse des mains. Rev. Neurol., **1:** 427–450, 1926.
25. Sainton, Paul: Contribution à l'étude anatomo-pathologique et clinique de l'amyotrophie Charcot-Marie. Nouv. Iconog. de la Salpêtrière, **12:** 206–215, 317–326, 1899.
26. Schneider, D. E., and Abeles, M. M.: Charcot-Marie-Tooth Disease with Primary Optic Atrophy. Report of Two Cases Occurring in Brothers. J. Nerv. and Mental Dis., **85:** 541–547, 1937.
27. Schwartz, L. A.: Clinical, Histopathological and Inheritance Factors in Peroneal Muscular Atrophy (Charcot-Marie-Tooth Type). J. Michigan Med. Soc., **43:** 219–230, 1944.
28. Siemerling, E.: Zur Lehre der spinalen neuritischen Muskelatrophie (Atrophia muscularis progressiva spinalis neuritica Bernhardt), (progressiven neurotischen oder neuralen Muskelatrophie Hoffmann). Arch. f. Psychiat., **31:** 105–127, 1898.
29. Symonds, C. P., and Shaw, M. E.: Familial Claw-foot with Absent Tendon-Jerks: A "Forme Fruste" of the Charcot-Marie-Tooth Disease. Brain, **49:** 387–403, 1926.
30. Taylor, James: ? Peroneal Atrophy. Proc. Royal Soc. Med., **6** (Neurological Section): 50, 1912.
31. Tooth, H. H.: The Peroneal Type of Progressive Muscular Atrophy. London, H. K. Lewis, 1886.

DISCUSSION

Dr. H. R. McCarroll, St. Louis, Missouri: I have heard these two excellent papers for the first time, and have had no opportunity to study them previously.

Dr. Patterson has made an excellent presentation, based on sound principles and sound treatment. There are, however, two minor points of difference in surgical technique and postoperative care which might be mentioned. One is the type of approach which is used for the stabilization. There is no reason to use a wide, sweeping incision that completely encircles the lateral malleolus. An adequate triple arthrodesis can be carried out through a relatively small incision, placed directly over the sinus tarsi. This decreases considerably the amount of postoperative difficulty.

Second, skin sloughing after triple arthrodesis or after panastragalar fusion can be diminished or avoided if the plaster cast is split postoperatively, in order to permit swelling and to avoid pressure necrosis of the overlying skin edges.

Dr. Patterson has done arthrodeses in twenty-two cases of Friedreich's ataxia. We have never done a stabilization in Friedreich's ataxia, and I would consider it contra-indicated. By removing the twenty-two arthrodeses in Friedreich's ataxia from his series, his percentage of good results is increased considerably. In the recurrent deformities, he made little or no mention of correction of muscle imbalance. A few years ago, we reported a series of 1,100 consecutive stabilizations for poliomyelitis, in which the highest percentage of recurrent deformity resulted from uncorrected muscle imbalance. Any paper dealing with stabilization of the foot is, in my opinion, incomplete unless that point is brought out.

The paper presented by Dr. Jacobs is also sound, but there are again a few points of difference that should be brought out. First, the stabilization, regardless of whose name is attached to it, is a modification of the triple arthrodesis, unless it is a subtalar or panastragalar fusion. In a Charcot-Marie-Tooth type of muscular dystrophy, we have weakness of the peroneal group of muscles and, at times, of all the dorsiflexors of the foot. Since we are dealing with a primary equinovarus deformity of the foot, we should, by all means, perform a Lambrinudi type of stabilization rather than a simple arthrodesis, as shown here.

Another important point is the selection of the proper time for corrective surgery in the Charcot-Marie-Tooth type of muscular dystrophy. If the process is progressive, stabilization should not be done. This, of all the progressive muscular atrophies, however, is notorious for reaching a stationary point. The individual should be watched, therefore, until a stationary point has been reached,—that is, until there has been no evidence of clinical progress in the muscle involvement for at least two years. Then the stabilization can be

(*Continued on page 47*)

FRACTURES OF THE LATERAL TIBIAL CONDYLE

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Fractures of the tibial condyles were brought into prominence in 1929 by the papers of Cotton and Berg, in Boston, and Cubbins, Conley, and Seiffert, in Chicago, one calling them "fender fractures" and the other "bumper fractures", because, as Cotton and Berg expressed it, they were so often caused by "the automobile in contact with the jay-walking citizen". Sever had already reported three fractures of the tibial condyle in 1916, and had discussed them again in 1922. Since then, a number of articles have been written and there is sufficient unanimity to establish the general principles governing the injury. The present paper is intended to review these general principles and to add a "therapeutic classification".

The basic mechanism for all fractures of the lateral tibial condyle is an impaction of the condyle of the femur against the plateau of the tibia, as a hammer strikes an anvil. Many authors have pointed out that three main variations can be differentiated. In a series of forty cases, the authors have classified these three general types. Type I, the "crush fracture", consists of all degrees of compression of the lateral tibial condyle with mild or extreme depression of the plateau. In Type II, or the "split fracture", the condyle, instead of being crushed, is split off with little, if any, depression. In Type III, or "shatter fractures", the force of the trauma extends obliquely toward the center of the knee and downward, to break away the support of the shaft or of the medial condyle at the neck of the tibia.

PATHOLOGY AND TREATMENT

Type I. Crush Fractures

The characteristic feature of this group of fractures (Fig. 1) is that the blow of the femoral condyle transmits its force directly downward on the tibia, thereby crushing the cancellous bone beneath the plateau. If the fibula remains intact, as usually happens, it will lend support to the plateau, which may be only slightly depressed; or again, as Barr suggests, the impacting force may create a bursting effect within the metaphysis which will fragment the trabeculations without greatly depressing them. If the fibula gives way, the entire lateral segment may slide downward *en masse*, causing a major displacement. If the blow of the femoral condyle strikes the rim of the plateau, the depression may be marginal; but frequently it tends to strike more medially, causing a severe central depression with comminution and impaction of articular cartilage. Thus, there may be marginal impaction, central impaction, displacement *en masse*, or any stage between.

In the splendid study of 125 cases, made over a six-year period by Cubbins, Conley, Callahan, and Scuderi, four variations of crush fractures were carefully described as Types 2, 3, 4, and 5. The essential problem of treatment in all of these cases is similar,—to elevate the plateau to a position as near normal as possible, and to maintain support long enough for solid healing of the crushed bone. No rule can be laid down for open or closed reduction, since the decision depends wholly upon the degree of displacement. Cotton's warning, based on the experience of years, deserves a hearing: "I am entirely against open reduction, and although I have done it often, I shall probably never do it again. Better reductions with more prompt healing and better final results can be obtained without open operation."

The success of closed reduction has been summarized by Bick, who studied a series of

sixty-one fractures. On the other hand, early surgical interference has been recommended for a large number of these cases by Cubbins and his associates[11], Leadbetter and Hand,

TABLE I

TYPE I — CRUSH FRACTURES

Case No.	Treatment	Meniscus Tear	Findings at Follow-Up				Length of Follow-Up (*Months*)
			Motion (*Degrees*)	Deformity	Pain*	Activity†	
1	Non-operative	No signs	70–180	0	Trace	Good	12
3	Non-operative	No signs	70–176	0	0	Excellent	18
4	Non-operative	No signs	Normal	0	0	Excellent	18
7	Operation refused	Probable	60–165	Extension block	Slight	Fair	18
8	Operation refused	Probable	80–165	Extension block	Moderate	Poor	16
9	Non-operative	No signs	65–180	0	Trace	Excellent	12
12	Non-operative	No signs	Patient failed to return; discharged with brace				1½
13	Non-operative	No signs	Patient failed to return; discharged with brace				2
14	Non-operative	No signs	Patient failed to return; discharged with brace				1
15	Non-operative	No signs	65–180	7 degrees of valgus	Trace	Good	12
16	Non-operative	No signs	Normal	0	0	Excellent	12
17	Patient aged 82; operation not advised	Probable	50–168	Extension block	Slight	Fair	12
18	Non-operative	No signs	58–180	0	0	Excellent	12
19	Non-operative	No signs	Did not return to follow-up clinic				
20	Non-operative	No signs	70–180	6 degrees of valgus	Slight	Good	12
23	Non-operative	No signs	58–180	0	Slight	Good	6
24	Non-operative	No signs	Did not return to follow-up clinic				
25	Non-operative	No signs	85–176	0	Slight	Good	6
28	Non-operative	No signs	Did not return to follow-up clinic				2
29	Non-operative	No signs	60–180	4 degrees of valgus	Slight	Good	12
30	Non-operative	No signs	60–180	0	Trace	Excellent	12
31	Non-operative	No signs	55–180	0	0	Excellent	4
32	Non-operative	No signs	80–180	0	Trace	Excellent	6
34	Non-operative	No signs	75–180	0	Slight	Good	12
35	Non-operative	No signs	Normal	0	0	Excellent	6
36	Non-operative	No signs	Normal	0	0	Excellent	6
37	Non-operative	No signs	72–180	0	Moderate	Poor	8
38	Non-operative	No signs	80–180	6 degrees of valgus	Slight	Good	6
39	Non-operative	No signs	75–180	0	Trace	Excellent	12

* The degree of pain is estimated as follows:

Trace: Mild pain after long or heavy exercise.

Slight: Slight pain in the course of normal activity, becoming severe after long or heavy exercise.

Moderate: Moderate pain with normal exercises,—enough to cause a limp and to prevent steady knee-straining exercise.

Severe: A disabling degree of pain except in very light exercise.

† The degree of activity is estimated as follows:

Excellent: Full resumption of normal activity, including usual knee-straining exercise.

Good: Ability to resume normal activity, but with definite limitation after prolonged knee-straining exercise.

Fair: Ability to resume light tasks, but limited in work involving any knee strain.

Poor: Inability to resume light tasks without difficulty.

Palmer, and others. Barr found that 50 per cent. of his cases had minimal displacement, not requiring surgery; 23 per cent. had slight to moderate displacement, where non-operative results seemed to be as good as those following operation; and 27 per cent. had moderate to marked displacement of fractures (over one-half inch of depression), requiring open reduction.

From this discussion, the points to emphasize are that the results of surgery are apt to be disappointing, and that, as stated by Bick, conservative treatment will give satisfactory results "even when considerable irregularity of the condylar tables persists". McBride, in discussing Barr's paper [2], remarked: "I have been surprised at the good functional result in several cases in which the reduction was very unsatisfactory from the x-ray standpoint". Maisel and Cornell have provided a satisfactory explanation for this discrepancy, and their point is significant. In reoperations upon patients previously treated for condylar fractures, they found: "The tibial articular surface was covered with a smooth, gray, shiny layer of fibrocartilage". This growth of fibrocartilage had restored

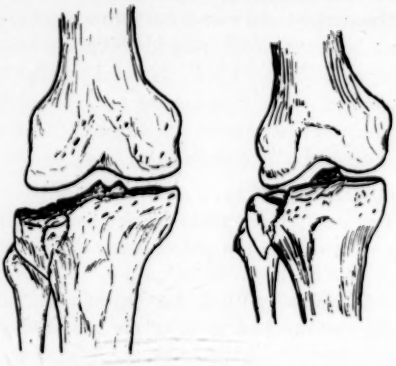


FIG. 1

Diagrams of two types of crush fractures.

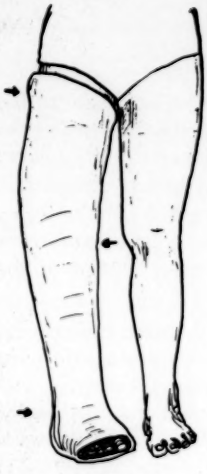


FIG. 2-A

Conservative treatment with a cast and brace.

FIG. 2-B

the plateau to nearly its normal level, although by roentgenogram the bone defect would naturally appear as serious as ever.

The principles of conservative treatment, followed by the authors, have been worked out in detail. First a manipulative reduction is done under anaesthesia. There is a similarity in the mechanism of this reduction and that of a compression fracture of the vertebra, where, as in the knee, spongy bone has been crushed, and where hyperextension of the compressed part restores its normal contours by the pull on the anterior ligament. The knee is drawn forcibly into varus and held with a well-applied cast, which must extend from the groin to the foot. Theoretically, the tibial collateral ligament will help to draw the depressed fragments back into place, but the results of this manoeuvre will sometimes be disappointing when judged by roentgenogram. Nevertheless, the clinical result is apt to be satisfactory if three points are observed: (1) The knee must be forced into enough varus to protect the lateral plateau from compression; (2) this position must be maintained for approximately four months; and (3) non-weight-bearing motion must be started early to avoid stiffness of the knee.

The authors' practice has been to split the cast as soon as primary repair has taken place,—that is, in ten days to two weeks. Moderate flexion movements are then started actively, but not passively. The exercises are repeated twice a day, the amount of motion being increased as fast as the patient can tolerate it. The limb is replaced in the cast after

TABLE II

TYPE II — SPLIT FRACTURES

Case No.	Treatment	Meniscus Tear	Findings at Follow-Up				Length of Follow-Up (*Months*)
			Motion (*Degrees*)	Deformity	Pain	Activity	
5	Non-operative	No signs	78–180	4 degrees of valgus	Slight	Good	12
6	Non-operative	No signs	Normal	0	Slight	Good	18
11	Operative	Lateral meniscus torn	50–180	0	Slight	Good	18
26	Non-operative	No signs	Normal	0	0	Excellent	12

exercise. Scudder has illustrated Cotton's suggestion that the patient lie on the unaffected side when doing the leg exercises, so that the weight of the foot and leg would produce varus at the knee. By the third or fourth week, the patient can usually regain a good range of motion without discomfort. Thereafter, he can be transferred from his bivalved plaster cast to a high-thigh, double-upright brace, equipped with straps to maintain varus support. Dobelle commented that no brace can maintain the bow-legged position permanently; but the authors find that, with a strap pulling outward at the knee and with an inward pull on the lower portion of the tibia, sufficient "varus support" can be maintained to relieve the lateral plateau from recompression (Fig. 2-B). The similarity to vertebral fractures is again strongly evident, for, as Böhler emphasized, vertebral fractures can be completely protected from recompression while the patients are doing active exercises in hyperextension casts.

The patient should wear his brace both night and day until the end of the twelfth week; but it should be removed at least twice a day for increasing periods of guarded knee-bending exercises. The most important elements in the exercise program are, of course, the patient's own cooperation and persistence. These are qualities which no surgeon can bestow, but every encouragement should be given. Lipscomb [1] has stated the point well: ". . . it behooves all of us who treat diseases and injuries of the knee to cajole, pamper, force or otherwise bring about the patients' co-operation".

The brace should be retained for all weight-bearing activity until solid bony healing has occurred. Some authors feel, as Bick does, that a brace may be discarded in six or seven weeks; but in the authors' experience, braces must be retained for at least three or four months unless the fracture is minimal. The trabeculations of spongy bone necessarily remain weak for months after the compression fracture, and it seems unreasonable to expect that this bone could support unguarded weight-bearing in less than four months.

In a certain number of crush fractures the damage is so extensive that operative treatment may be necessary; but the degree of crushing which demands open surgery is very much in debate. Cubbins and his associates [11] were among the earliest enthusiastic advocates of operation and stated: "we are certain that fully 40 per cent. [of all cases] could be markedly improved with an open operation. . . ." From personal experience, the authors feel that the percentage is less, although indications for open reduction must be recognized in cases of severe compression. For the surgical approach, the authors prefer Cave's method, which he describes as ". . . a bayonet incision, beginning just posterior to the lateral femoral epicondyle about 3 inches above the joint line and curving forward at the joint line, and down along the lateral portion of the tibial crest for 4 inches". After exposure of the fracture, the depressed plateau is elevated and supported in place by a

bone graft or a piece of bank bone. Palmer advises using spongy os purum, and he calls the repair a "reposition" and "plugging". Emphasis should be placed on the importance of the postoperative follow-up, along the same lines as that recommended for the non-operative cases. Without this convalescent care, good surgery will often be wasted.

Another indication for open surgery in crush fractures lies in displacement or tearing of the lateral meniscus. Some surgeons feel that routine exploration of the meniscus should be carried out. The authors disagree with such a policy, because they have found the percentage of tears of the lateral meniscus to be very small. This conservative attitude is supported by sound judgment, as well as clinical experience.

Cotton protested against routine removal, since the meniscus helps to deepen the articulation for the femoral condyle; Clarke commented: "it seems particularly undesirable to remove the meniscus. . . ." The authors believe that clinical symptoms give sufficient evidence of a displaced cartilage, and that no exploration should be carried out unless positive indications are present.

In the present series of twenty-nine Type I fractures, clinical signs of cartilage injury developed in only three cases. These patients showed loss of over 10 degrees of full extension, and one of them complained of a painful clicking sensation. None of these patients could be operated upon, however. One was an alcoholic who failed to return; one was a busy housewife who had no serious pain, despite her loss of extension, and refused operation; and one was a lady of eighty-two years, whose clinical result was so satisfactory that the authors did not urge operation. This experience illustrates the point stated by Barr, who felt that, even though the meniscus may usually be torn enough to be partially displaced, ". . . it rarely, if ever, produces symptoms due to locking".

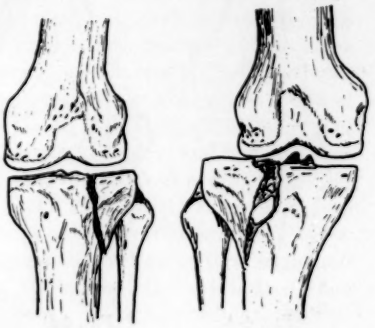


FIG. 3-A FIG. 3-B

Two types of split fractures.

TABLE III

TYPE III — SHATTER FRACTURES

Case No.	Treatment	Meniscus Tear	Findings at Follow-Up				Length of Follow-Up (*Months*)
			Motion (*Degrees*)	Deformity	Pain	Activity	
2	Operative	Lateral meniscus torn	78–180	4 degrees of valgus	Moderate	Good	18
10	Non-operative	No signs	55–180	0	Moderate	Fair	12
21	Operative	Both menisci torn	65–180	0	Slight	Good	14
22	Non-operative	No signs	55–180	0	Slight	Good	12
27	Operative	Lateral meniscus torn	75–173	0	Moderate	Poor	12
33	Non-operative	No signs	80–180	0	Moderate	Fair	16
40	Operative	Lateral meniscus torn	Sepsis developed at operation and wound is still draining.				8

Type II. Split Fractures

This type of fracture is derived from the same basic mechanism as Type I; but its character is wholly different, since it consists of a lateral displacement rather than downward crushing. A vertical line of separation will often appear, as clean-cut as if an ax had split a piece of kindling wood (Fig. 3-A). The problem here is merely to replace and hold the lateral fragment. Where displacement is slight, conservative treatment will undoubtedly serve.

For reduction of the moderately severe fractures various methods have been suggested, including use of the Forrester clamp or of Caldwell's "nut-cracker" lever. Metz and his co-workers used long pressure tongs. In the authors' experience, reduction has been relatively easy to accomplish, even with manual manipulation; but as soon as the compressing force has been released, the fragments spring apart again. To prevent this, Buckner introduced double wires with buttons on the ends, which he tightened with a special apparatus. The authors believe that, where the displacement is gross, a considerable advantage can be gained by open reduction and screw fixation. Ahern and Lipscomb, at the Mayo Clinic, recommended a Johansson lag screw, and other surgeons favor bolts; but the method of fixation is optional, as long as the fragment is held firmly in place. Frequently, a simple carpenter's screw will be adequate.

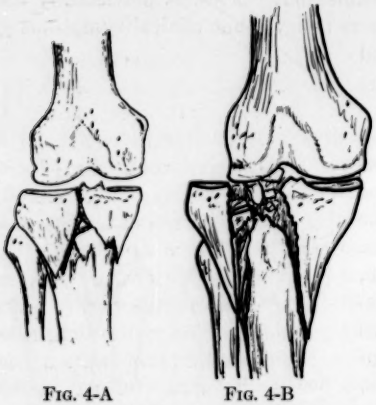


FIG. 4-A FIG. 4-B

Showing mild and severe types of shatter fractures.

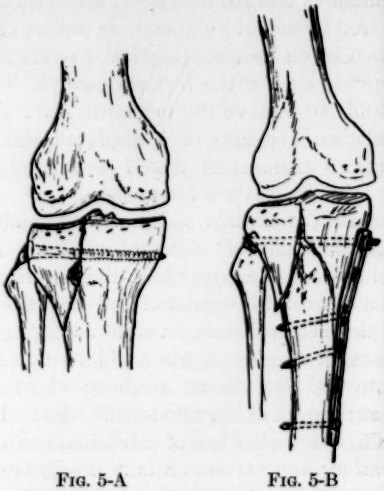


FIG. 5-A FIG. 5-B

Illustrating treatment by open reduction.

At the time of operation, it is important to explore the lateral meniscus, since there is a tendency for it to be displaced into the cleft. At operation in one of the authors' cases, the major portion of the meniscus was found to be deeply embedded between the fragments. This will be explained by recalling that the anterior and posterior attachments of the meniscus are fixed to the central portion of the tibial plateau, together with a posterior attachment which is carried upward, to be inserted into the medial condyle of the femur (ligament of Wrisberg). The lateral attachment of the meniscus is not inserted into bone, but to the inner surface of the joint capsule. Thus, when the fragment of tibial bone springs out laterally, the meniscus is tightened like a bow-string and dragged into the cleft of the fracture. Operative removal then becomes necessary, in order to reduce the fracture. Again, the importance of follow-up care of the type already outlined must be stressed. The final success of surgical treatment will depend upon early, guarded motion, and upon prolonged protection with a brace.

Type III. Shatter Fractures

These fractures are of the same type as those called "bursting" and "spreading" by other authors. The neck of the tibia is shattered and both the medial and lateral condyles

are split off (Figs. 4-A and 4-B). Sometimes the fracture line extends so far downward into the metaphysis that the medial cortex of the tibia is broken. Evidently the impacting force in these fractures is greater than that producing fractures of Type I, and probably the femoral condyle is aimed a little more obliquely in the medial direction. Obviously this injury leaves no solid support to receive the weight transmitted by the femur on either tibial plateau. Where marked displacement has occurred, there is a high probability of damage to the lateral meniscus. In one of the authors' cases, even the medial meniscus was completely displaced, due to the wide medial displacement of the medial tibial condyle.

As in the other categories, no arbitrary rule can be made to distinguish between operative and non-operative cases. Attention must be given to Cave's emphatic statement regarding these fractures: "Open reduction is contraindicated." Even the best operation will not in itself greatly shorten the convalescence, and, furthermore, the operative procedure is so extensive that it involves more than the ordinary risk of infection. The literature is remarkably free from any mention of this hazard; but, even with antibiotics and chemotherapy, the risk remains grave. The authors, therefore, practise conservative treatment wherever there is a reasonable expectation of success. In shatter fractures with linear displacement, the limb should be placed in a cast and the patient should be followed on a program similar to that indicated for fractures of Type I. Of course, varus support will not be needed, inasmuch as the whole stability of the knee is gone, and not just that of the lateral condyle. Braces should be used to support the knee in neutral position; weight-bearing, even in the brace, should be prohibited until very late. In the more severe shatter fractures which are too comminuted for the bone to hold fixation screws or bolts, the authors have used Patterson's adaptation of the suspension-traction method suggested by Caldwell. This includes an upper-thigh cast and a Thomas splint, with hinged knee action through Pearson attachments. After four to six weeks, this apparatus may be discarded in favor of a brace and crutches, but the patient should be urged to carry out bent-knee exercises frequently throughout the day. Convalescence will take seven months or more in severe cases.

If the displacement is gross, with fragments large enough to permit some form of internal fixation, the advantages of open reduction probably outweigh the risks of surgery. Simple bone screws in the tibial cortex, with a bolt through the condyles, may afford sufficient fixation. In the really unstable tibial shafts, however, the authors have found it convenient to employ a Townsend and Gilfillan plate which can be screwed to the tibial shaft and can extend upward to receive a bolt transfixing the upper condylar fragments, thus fastening all the fracture surfaces into a unified mechanism of internal fixation (Fig. 5-B). The authors' approach has been through a bayonet-shaped incision, beginning lateral to the lower portion of the patella and extending vertically downward to the level of the plateau, then curving sharply across the front of the tibia just below the tubercle and extending down the medial side of the crest of the tibia for four inches. Retraction of the skin flaps allows very wide exposure through this approach, and adequate exposure is essential to the procedure.

The knee joint must be thoroughly explored; this can be done by opening the capsule both laterally and medially to the patellar tendon. Cubbins, Conley, Callahan, and Scuderi warned: "Small fragments of bone and cartilage are often encountered in the joint fluid, and we have expressed several from out of the suprapatellar pouch". If the meniscus is torn, it should be removed; and, if there is a large crush defect in the plateau, an iliac graft may be used to fill the cavity. The anatomical results of this operation are apt to be very satisfactory; but the clinical end result will be uncertain for four to six months after operation.

The authors have used this type of operation in four patients, but it is still too early to evaluate the long-range results. In two cases the results have been very successful, although one patient's convalescence was so slow as to be discouraging at first. A third

case was more disappointing, due to the development of a pressure sore over the line of incision. This required skin-grafting, which further delayed rehabilitation exercises. There now promises to be a fair end result, the knee having a range of motion of 75 to 173 degrees (Case 27). In the fourth patient (Case 40) massive infection developed and the result is, therefore, a failure.

Throughout the postoperative stage, everything depends upon strengthening the leg muscles to support the knee, and consequently the patient's self-rehabilitation is very important. It has been suggested that patients of the non-cooperative type should be treated by fusion operation at the start. This seems like admitting failure too early, and the authors feel strongly that fusion ought to be reserved as a last resort in the very worst cases.

RESULTS OF FOLLOW-UP STUDY

Tables I, II, and III present the follow-up results seen after careful study.* Although the statistical charts speak for themselves, a few points deserve mention:

1. Type I fractures occur more than twice as often as the other two types combined,—the latter being about equally divided.

2. Although not tabulated, it might be mentioned that 57 per cent. of all fractures were caused by falls, either down stairs or other falls; 33 per cent. were produced by automobile bumpers or other external blows against the knee; and 10 per cent. resulted from unknown causes.

3. Comparison of the three types of fractures shows the value of this classification from the standpoint of operative indications, pathological interpretation, and prognostic outlook. The eventual outcome is much worse in Type III than in the other two types; a greater amount of surgery is required and there is a far greater proportion of torn and displaced menisci.

4. The authors do not consider their conservative attitude toward Type I fractures to be conclusive, and recognize that it disagrees with the reports of Cubbins and his associates, based on wider experience. Nevertheless, in this series very little indication was found for open reduction of crush fractures. Conservative treatment must be carried out under constant and careful supervision, with emphasis on prolonged protection combined with early and active motion. The results of this method, as seen, are good.

* The authors are indebted to their former chief, Otto J. Hermann, M.D., who served as impartial observer in helping to evaluate a series of these cases after twelve months.

REFERENCES

1. Ahern, G. S., and Lipscomb, P. R.: Fracture of the Tibial Plateau: Report of Case. Proc. Staff Meet., Mayo Clin., **23**: 288–290, 1948.
2. Barr, J. S.: The Treatment of Fracture of the External Tibial Condyle (Bumper Fracture). J. Am. Med. Assn., **115**: 1683–1687, 1940.
3. Bick, E. M.: Fractures of the Tibial Condyles. J. Bone and Joint Surg., **39**: 102–108, Jan. 1941.
4. Böhler, Lorenz: Treatment of Fractures, Fourth English Ed., p. 129. (Translated by E. W. Hey Groves.) Baltimore, William Wood and Co., 1935.
5. Buckner, H. T.: Bumper Fractures of the Tibia. Northwest. Med., **37**: 102–105, 1938.
6. Caldwell, E. H.: Fractures of the Condyles of the Tibia. Surg., Gynec., and Obstet., **63**: 518–522, 1936.
7. Cave, E. F.: Fractures of the Tibial Condyles Involving the Knee Joint. Surg., Gynec., and Obstet., **86**: 289–294, 1948.
8. Clarke, H. O.: Discussion on Fracture of the Tibia Involving the Knee-Joint. Proc. Royal Soc. Med., **28**: 1035–1043, 1935.
9. Cotton, F. J.: Fender Fractures. Surg., Gynec., and Obstet., **62**: 442–443, 1936.
10. Cotton, F. J., and Berg, Richard: "Fender Fracture" of the Tibia at the Knee. New England J. Med., **201**: 989–995, 1929.
11. Cubbins, W. R.; Conley, A. H.; Callahan, J. J.; and Scuderi, C. S.: Fractures of the Lateral Condyle of the Tibia. Classification, Pathology, and Treatment. Surg., Gynec., and Obstet., **59**: 461–468, 1934.

12. Cubbins, W. R.; Conley, A. H.; and Seiffert, G. S.: Fractures of the Lateral Tuberosity of the Tibia with Displacements of the Lateral Meniscus between the Fragments. Surg., Gynec., and Obstet., **48:** 106–108, 1929.
13. Dobelle, Martin: A New Method of Closed Reduction of Fracture of the Lateral Condyle of the Tibia. Am. J. Surg., **53:** 460–462, 1941.
14. Leadbetter, G. W., and Hand, F. M.: Fractures of the Tibial Plateau. J. Bone and Joint Surg., **22:** 559–568, July 1940.
15. Maisel, Bernard, and Cornell, N. W.: Conservative Treatment of Fractures of the Tibial Condyles. Surgery, **23:** 591–598, 1948.
16. Metz, A. R.; Householder, Raymond; and DePree, J. F.: Impaction of Fractures by Large Pressure Tongs. Am. J. Surg., **59:** 447–449, 1943.
17. Palmer, Ivar: Compression Fractures of the Lateral Tibial Condyle and Their Treatment. J. Bone and Joint Surg., **21:** 674–680, July 1939.
18. Patterson, R. E.: Personal communication.
19. Scudder, C. L.: The Treatment of Fractures, Ed. 11, p. 973. Philadelphia, W. B. Saunders Co., 1939.
20. Sever, J. W.: Fracture of Tuberosities of the Tibia. A Report of Three Cases. Am. J. Orthop. Surg., **14:** 299–302, May 1916.
21. Sever, J. W.: Fractures of the Tibial Spine Combined with Fractures of the Tuberosities of the Tibia. Surg., Gynec., and Obstet., **35:** 558–564, 1922.

DISCUSSION

Progressive Muscular Atrophy of the Peroneal Type

(*Continued from page 38*)

carried out. It should always be followed by muscle transplantation in an attempt to re-establish a balance of muscle power on the two sides of the foot. The balance of muscle power is important, although if we do a Lambrinudi type of stabilization, its importance is diminished.

Tenotomy of the heel cord, which was mentioned as a subcutaneous tenotomy, is a dangerous procedure. Why take a chance of destroying the little bit of muscle power left in this foot? By all means, do an open lengthening of the heel cord and preserve the calf group, because these are the muscles through which this individual will be able to retain what little function he has.

Dr. William M. Roberts, Gastonia, North Carolina: It has been my pleasure to read both of these papers, and I appreciate the great effort that these authors have made.

In Dr. Jacob's paper the term "Lambrinudi" interested me, because Neal Garber came back from England and he was describing this "Lambrinudi" operation, which we call a Hoke stabilization. I would like to ask our Nomenclature Committee, if possible, to get some one standard term applied to triple arthrodesis. Those of us who were exposed to the teachings of Dr. Hoke cling to the name Hoke stabilization, and yet few today perform a typical Hoke stabilization; all of us fuse the calcaneocuboid joint, I am sure. This joint is always included in the stabilization operation at our institution; we feel it is one of the most important joints for fusion. A review of Dr. Patterson's paper will bear out that, unless this joint is included in the fusion, the good results will be fewer.

Like Dr. McCarroll, I want to make a plea for the short stabilization approach. All of us who came under the teachings of Hoke carry out stabilization of the foot through a much shorter incision than that advocated by Dr. Patterson. Our incision usually starts just lateral to the head of the talus and extends posteriorly in the direction of the mid-point of the calcaneus. We visualize the extensor tendons anteriorly and the peroneals posteriorly. We do not open the peroneal sheath unless we are going to use the peroneal for a transplant, and neither do we cut the fibulocalcaneal ligament. I believe this incision gives a perfectly good exposure.

I would like to mention one instrument used in stabilization,—namely, the Hatt spoon. Among Dr. Hoke's original instruments was a spoon that he used to sweep around between the head of the talus and the navicular, and this was improved by Dr. Hatt.

Another point in technique that I would like to mention, particularly to the younger group, is the care in going through the sinus tarsi, particularly in dissecting the sinus tarsi pad in a valgus foot. When stabilization has been completed, a valgus foot will be taut on the lateral aspect, and if the surgeon goes through the fat pad and the sinus tarsi and sweeps it out, it will be very difficult to suture the skin edges. I think this is one of the most common reasons why this particular incision breaks down. I still feel that ample exposure can be obtained through this relatively short incision.

ARTHRODESIS OF THE HIP JOINT

A Follow-up Study *

BY FRANK E. STINCHFIELD, M.D., AND WILLIAM U. CAVALLARO, M.D., NEW YORK, N. Y.

This follow-up study presents a report of 117 patients (fifty males and sixty-seven females) upon whom hip-joint arthrodesis was performed for conditions other than tuberculosis, at the New York Orthopaedic Dispensary and Hospital, during the period from 1935 to 1946, inclusive. Each case was analyzed carefully as to symptoms, physical findings, type of surgery, and progress. To complete the study, the patients were called in recently for an interview and examination; check roentgenograms were taken when indicated. No patient who could not be located for follow-up examination at the end of one year has been included in the evaluation. If solid fusion was not obtained at the end of twenty-four months, the case is considered a failure, even though ultimate fusion may have developed. The follow-up period ranged from two to thirteen years, with an average of seven years.

The operative procedures included the trochanteric graft (Hibbs' type of fusion), the iliac graft, the Watson-Jones type, intra-articular fusion (with or without straight nails), and combinations and variations of these methods. All patients in whom solid bony arthrodesis developed were free of hip pain. The average positions of arthrodesis were flexion of 35 degrees, abduction of 10 degrees, and external rotation of 15 degrees. Arthrodesis with about 30 degrees of flexion seemed to give the patients the most comfort. Greater flexion improved the ability to sit, but made walking more difficult; the reverse was true when arthrodesis was done in a position of less flexion.

Postoperative treatment consisted of the following regimen, the periods of time listed being the average in each instance: rest in bed in a spica for fourteen weeks, ambulation in a spica for eleven weeks, and walking with crutches for three weeks; walking without support was permitted in twenty-eight weeks. If a revision of the arthrodesis was required, the method usually consisted of insertion of a Smith-Petersen nail, if no internal fixation had been used, and packing the area with bone chips.

For discussion purposes, the cases are divided into groups according to diagnosis, and include (1) poliomyelitis with dislocation, (2) congenital dislocation, (3) suppurative arthritis, and (4) osteo-arthritis. The etiology in the last group included coxa plana, fracture of the femoral neck, slipped femoral epiphysis, traumatic dislocation, multiple joints with osteo-arthritis, central dislocation, osteochrondritis dissecans, and Charcot's joint.

The purpose of this paper is not to evaluate arthrodesis as a method of treatment, but rather to present an objective analysis of a group of cases in which it was used. The findings obtained in the various etiological groups are submitted:

Poliomyelitis with Dislocation

Nine patients required hip-joint arthrodesis because of poliomyelitis; two were males and seven were females. The primary indication for surgery was instability rather than pain, although pain was severe in three cases. All of the patients lived a sedentary existence; seven required crutches at all times, and two used crutches when outside the house. All had severe paralysis of at least one lower extremity and all had had acute poliomyelitis in infancy, the average age at onset being 1.5 years. Pain and dislocation did not occur until about two years prior to arthrodesis; this was done at an average age of 22.5 years,

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 26, 1949.

the extremes being thirteen and thirty-one years. Restriction of activity in this group was severe, none of the patients being able to walk more than four blocks.

Evaluation of the hip *per se* was difficult in this group because of the degree of paralysis, which frequently necessitated other operative procedures involving the lower extremity. None of the patients had had previous hip surgery. Most of them were markedly improved after operation and led moderately active lives, seven of the nine discarding crutches entirely.

Congenital Dislocation

Twenty patients were operated upon for congenital dislocation of the hip. Because of a wide variation in ages, this group was analyzed in two sections,—those under twenty-one and those over thirty-five. No cases occurred between these ages, and an average would not give a true picture.

There were eleven patients in the younger age group, two males and nine females; 91 per cent. had had previous hip surgery, including four open reductions, two closed reductions, and five shelf stabilizations. Two patients had had two procedures. The chief complaints at the time of arthrodesis were limp, pain, fatigue, and deformity in ten instances, and stiffness in malposition in one. Following successful arthrodesis, the patients in this group were satisfied with their ability to complete their daily physical requirements. The average age at the time of arthrodesis was 15.5 years; the youngest patient was ten years old.

Nine patients with congenital dislocations, one male and eight females, had arthrodesis after the age of thirty-five, the oldest being forty-nine. Five had bilateral dislocation, and all had pain and deformity as the presenting symptoms. Five had had previous hip surgery: Two had closed reductions, one had a Lorenz osteotomy at the age of forty-seven, and two had soft-tissue operations. Better results were obtained in the younger group than in those who had arthrodesis after the age of thirty-five.

Suppurative Arthritis

In this group there were nineteen patients, nine males and ten females. The age at onset of the acute disease ranged from one month to twenty-six years, the average being six years. Pain and restriction of activity were the chief complaints. One-third of the group had had previous surgery, including one open and one closed reduction, two shelf stabilizations, and two reconstructions. Following arthrodesis, the patients in this group were able to participate in competitive athletics. The average age at the time of operation was sixteen years; the youngest was three and the oldest, thirty-one.

Osteo-Arthritis

Sixty-nine patients (thirty-three males and thirty-six females) were operated upon for osteo-arthritis of the hip. Pain was the presenting symptom in sixty-one instances, with an average duration of six years. Eight patients had bilateral involvement. The average age at the time of arthrodesis was forty-five, ranging from fifteen to seventy years.

In this group, seven patients had solid union at the end of one year; but they have been lost to follow-up and so are not included in the evaluation. Four, ranging in age from fifty-one to sixty years, died of pulmonary embolism or coronary occlusion between the first and third weeks. Of the remaining fifty-eight patients, solid bony union occurred in forty-three. In ten instances, union was delayed,—at the femur in three cases, and at the ilium or across the joint in seven. However, in all, progress to solid union took place in less than a year. Where arthrodesis was successful, complaints referable to the hip were absent and activities depended upon the general physical condition, arthritis of other joints, age, and the physical requirements of the patient.

Chart I

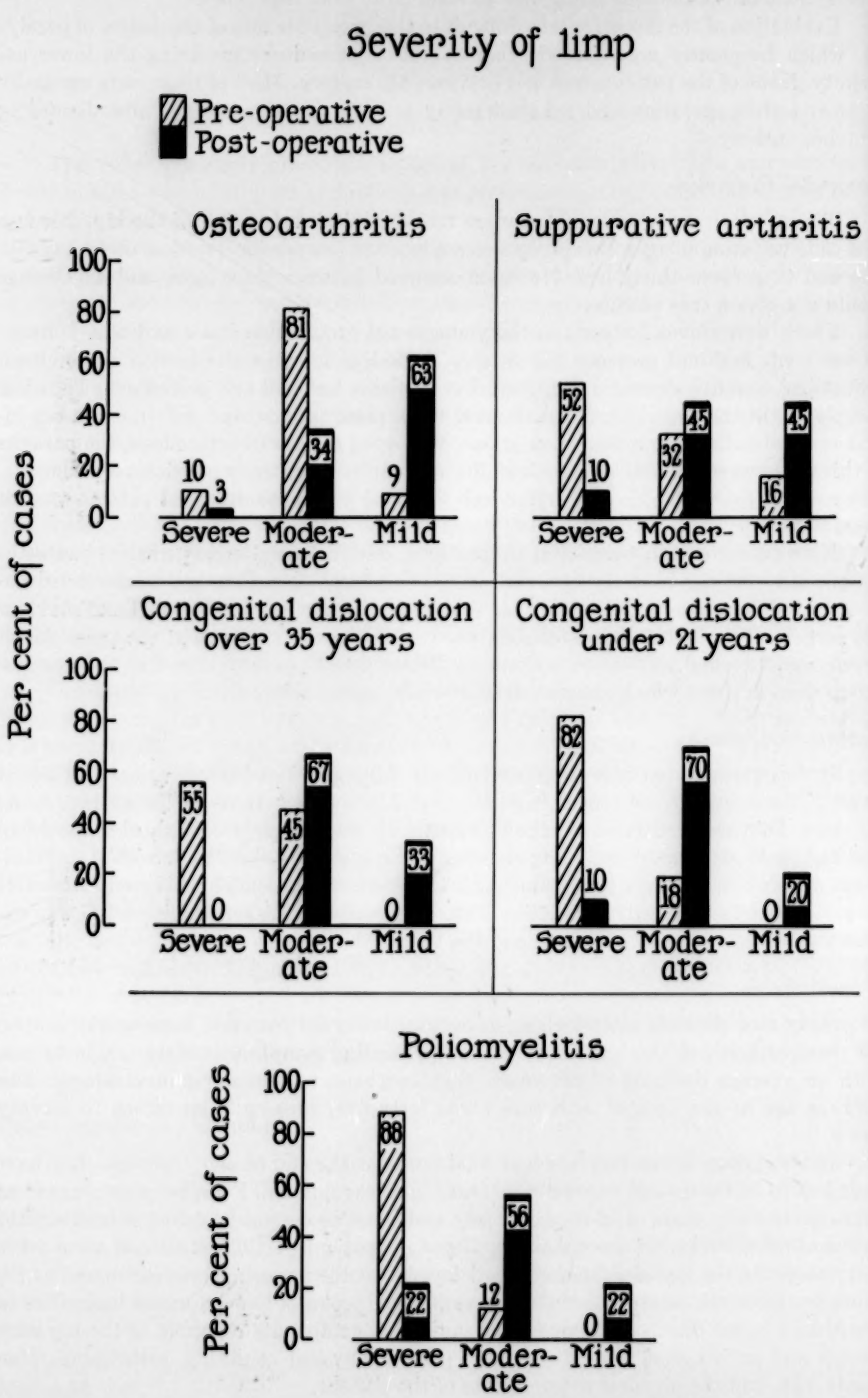

Severity of limp
Pre-operative
Post-operative
Per cent of cases
Osteoarthritis
100
80
60
40
20
0
10
3
81
34
9
63
Severe
Moder-ate
Mild
Suppurative arthritis
52
10
32
45
16
45
Severe
Moder-ate
Mild
Congenital dislocation over 35 years
55
0
45
67
0
33
Severe
Moder-ate
Mild
Congenital dislocation under 21 years
82
10
18
70
0
20
Severe
Moder-ate
Mild
Poliomyelitis
88
22
12
56
0
22
Severe
Moder-ate
Mild

Chart II

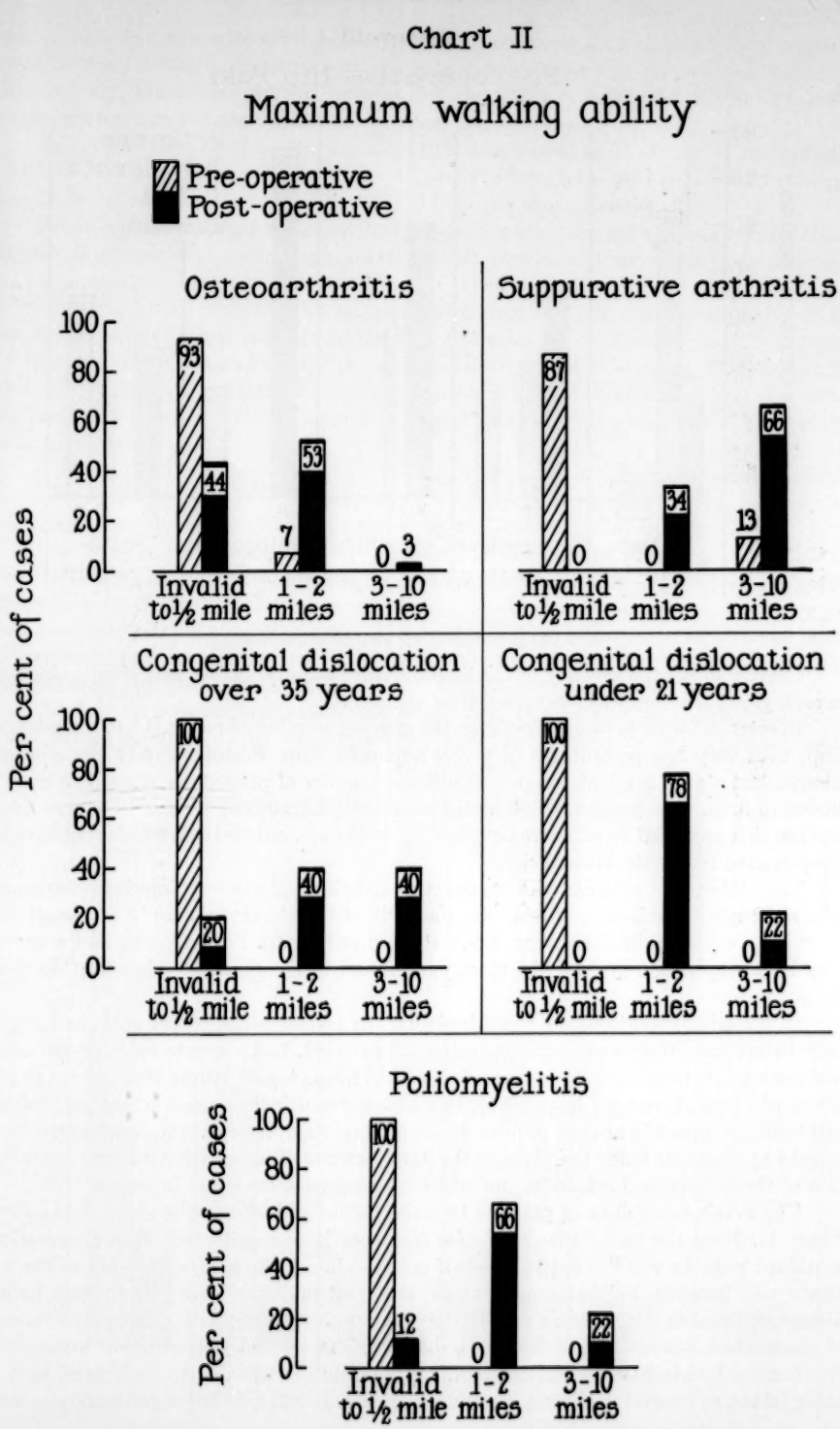
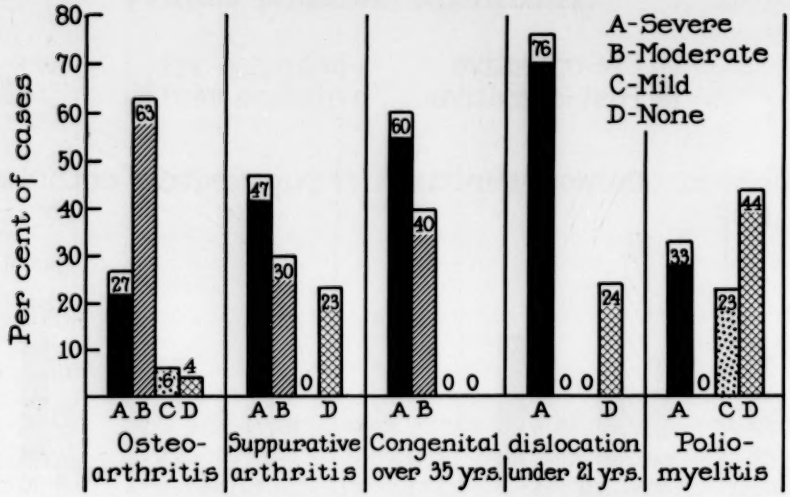

Maximum walking ability
Pre-operative
Post-operative
Per cent of cases
Osteoarthritis
93
44
7
53
0
3
Suppurative arthritis
87
0
0
34
13
66
Congenital dislocation over 35 years
100
20
0
40
0
40
Congenital dislocation under 21 years
100
0
0
78
0
22
Poliomyelitis
100
12
0
66
0
22
Invalid
to ½ mile
1-2 miles
3-10 miles
0
20
40
60
80
100

Chart III

Pre-operative Hip Pain



ANALYSIS OF CHARTS AND DISCUSSION

Chart I shows the severity of the limp in each category, before and after operation. In each group the limp was less severe after operation.

In osteo-arthritis before arthrodesis, the greatest number of patients had a moderate limp, with very low percentages of severe and mild limp. Postoperatively the greatest number had a mild limp, although a significant number of patients continued to have a moderate limp. The position of abduction with equal limb lengths caused severe limp, because this produced an effective lengthening on the arthrodesed side which could not be compensated for by the normal side.

The majority of patients with suppurative arthritis had a severe limp before arthrodesis, although a significant number are found in the moderate column. Postoperatively there is an equal division between moderate and mild limp. Patients who had a severe limp before operation are, for the most part, found in the moderate classification after operation.

In the other conditions the trend is similar. In poliomyelitis, 88 per cent. had severe limp before operation; while after operation 22 per cent. had a severe limp, 56 per cent. had a moderate limp, and 22 per cent. had a mild limp. A severe limp was present in patients who had shortening in excess of two and one-half inches, upon whom arthrodesis had been performed in neutral position or slight abduction. Attempts to equalize the limb lengths by shoe lifts failed to eliminate the limp, because of the relative increase in abduction of the arthrodesed extremity, for which no compensation could be made.

The maximum ability of patients to walk, before and after arthrodesis, is shown in Chart II. Here the trend toward longer distances is demonstrated. Before operation, sustained walking was limited to one-half mile in almost all cases, and many of the patients were invalids. Following arthrodesis, the great majority were able to walk longer distances. Invalids were present only in the group with osteo-arthritis due to involvement of other joints. Except in rare instances, the distance a patient walked before arthrodesis was limited by hip-joint pain. Postoperatively, limitation was due to back pain, pain in other joints, or general debility of the older patients. It was noted that patients who were

able to walk one mile without resting were able to complete their daily physical requirements without difficulty. Patients with all types of occupation were represented, including office worker, housewife, student, printer, laborer, janitor, bartender, laboratory technician, porter, telephone-switchboard operator, and dishwasher.

Agility and the ability to run depended to a large extent upon the age of the patient. Children were able to run with the greatest facility; adults were usually loathe to attempt this degree of exertion, but generally were able to run short distances.

Patients in the younger age groups participated in athletics, such as baseball, football, softball, tennis, skating, swimming, and basketball. One patient ran a mile in six and one-half minutes; one was inducted into the Army and served in Hawaii. Many between twenty-five and fifty years of age engaged in swimming and golf, one was an active scout leader, and several others enjoyed hiking as a diversion.

Stair-climbing was awkward, but was done effectively in most cases. The position of abduction caused most of the difficulty, and those best able to climb stairs "one over one" had had arthrodesis in 35 to 45 degrees of flexion. Stair-climbing one step at a time was more satisfactory.

Even among those who were more athletically inclined or who enjoyed hiking, walking downhill caused an increase in the severity of the limp and a slowing of pace. High chairs and stools were almost universally preferred to low, soft chairs for sitting.

The severity of preoperative hip pain among the various groups is compared in Chart III. The reason for arthrodesis in most cases of osteo-arthritis, suppurative arthritis, and congenital dislocation of the hip was the degree of pain. In patients with little or no pain, marked deformity was the main factor necessitating arthrodesis. In patients with poliomyelitis, pain was less severe, so that instability and the desire to be free of braces or crutches were the chief considerations.

The over-all pseudarthrosis rate was 23 per cent. (Chart IV). In the osteo-arthritis group, fifteen patients (22 per cent.) failed to attain solid arthrodesis on the first attempt.

Chart IV

Failures in Relation to Condition

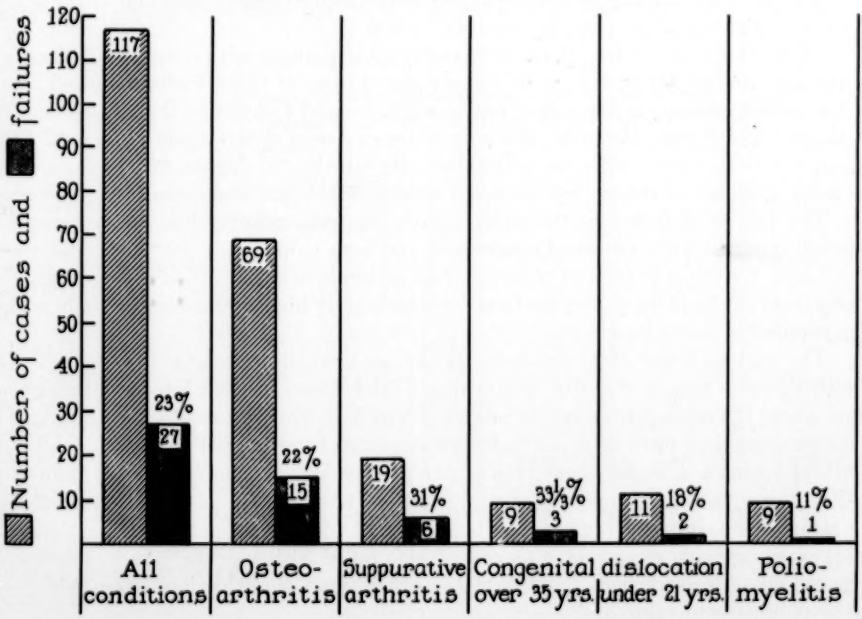
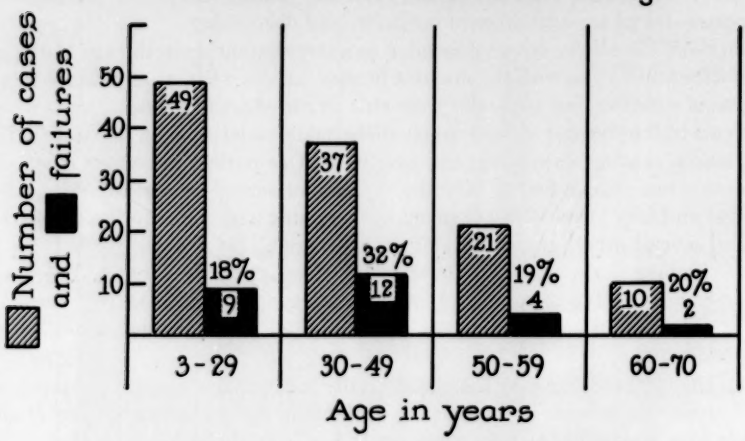


Chart V

Failures in Relation to Age



Three of these were not followed up; of the remaining twelve, five had eventual fusion after repair of the pseudarthrosis. In one of the failures there was no evidence of union after four years; but, without further surgery, solid bony fusion developed by the eighth postoperative year.

Six patients (31 per cent.) of those with suppurative arthritis failed to obtain arthrodesis, although fusion resulted after a secondary procedure in three of them.

In patients over thirty-five years of age with congenital dislocation of the hip, 33 per cent. of the operations failed. One was a Brittain type of arthrodesis in which there was non-union at the osteotomy site after two years. Another was a case of gross infection; this patient has subsequently been treated elsewhere with ischiofemoral transplants, without success. The third case was an intra-articular fusion with a three-flanged nail; this resulted in reduced motion, slight pain, but no bony fusion.

There were two failures (18 per cent.) among the patients with congenital dislocation of the hip, treated before the age of twenty-one. In one of these, fusion followed repair of the pseudarthrosis; in the other, repair was done and fusion was thought to be solid at the end of one year. However, this patient was not seen at the Clinic again until 1948, which was twelve years after the arthrodesis. He now has 50 degrees of free motion and no x-ray evidence of fusion, but he is not incapacitated and wishes no further surgery.

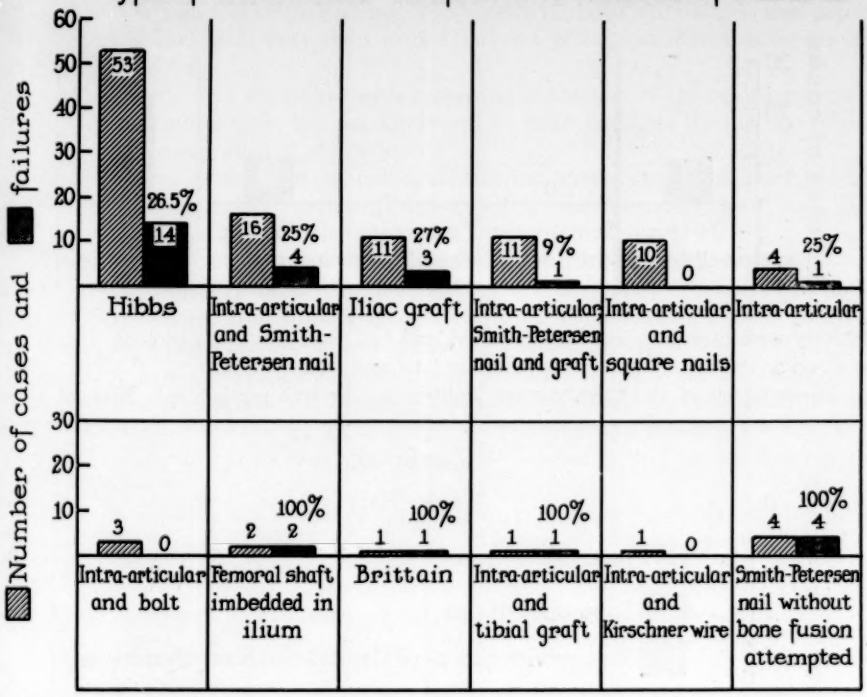
The failure of fusion in the patient suffering from poliomyelitis was successfully treated by repair with a Smith-Petersen nail and bone chips.

Chart V shows the failures resulting from arthrodesis in the various age groups. The group from thirty to forty-nine contained a significantly higher percentage of failure than the younger or the older groups.

The various types of arthrodesis—Hibbs' method, intra-articular fusion with the Smith-Petersen nail, iliac grafts, and intra-articular fusion without internal fixation—all show about the same percentage of failures (Chart VI). The best results were obtained in patients treated by intra-articular fusion accompanied by a Smith-Petersen nail and bone graft, or by intra-articular fusion plus carpenter-type Vitallium nails, inserted from three different directions to stabilize in several planes of motion. The poorest results followed an attempt to arthrodese the hip joint by the use of a long three-flanged nail without the joint cartilage being removed. In all cases fusion failed, although the pain was decreased appreciably. An insufficient number of the other types of arthrodeses were performed to be of statistical value.

Chart VI

Type of Arthrodesis Compared to Number of Failures



Of the twenty-seven failures in this series, ten patients did not have surgical repair. Four of these, who had non-union at the end of two years, progressed to solid union with weight-bearing between two and three years. In four other cases, the patients were

Chart VII

Location of Pseudoarthrosis in cases where Bone Grafts Were Used

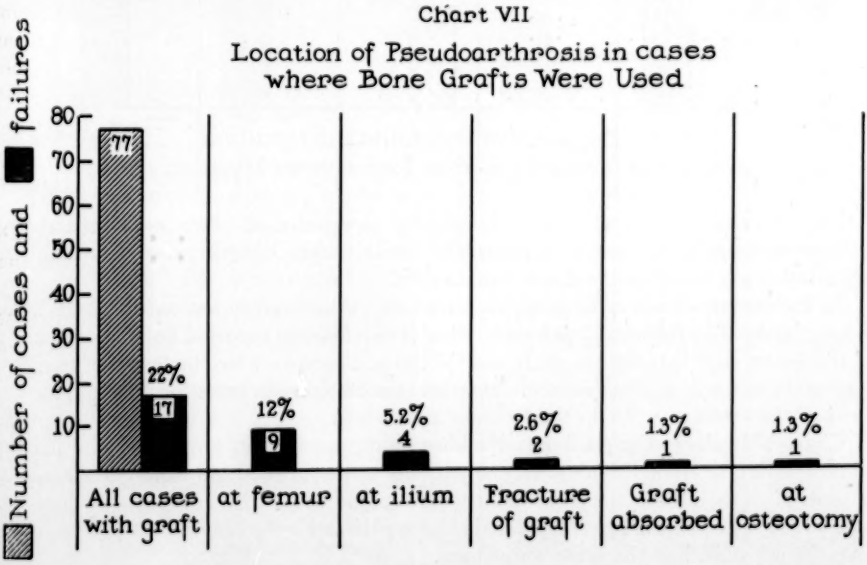
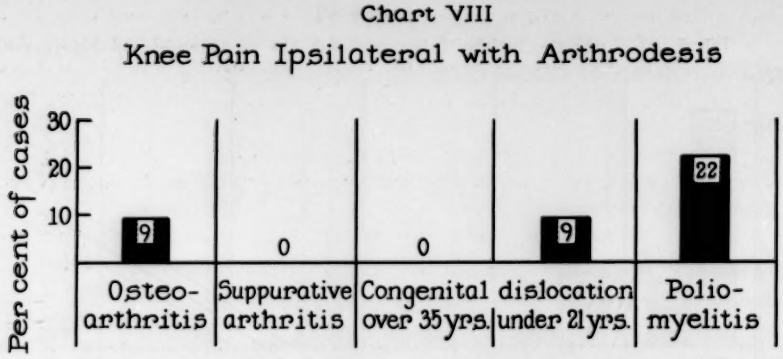


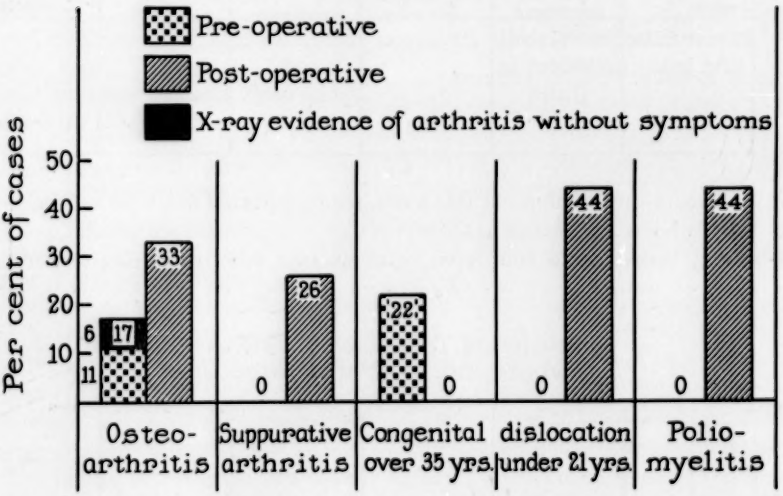
Chart VIII



relatively asymptomatic, not incapacitated, and had pain only with prolonged standing or excessive walking. Two were invalids, but refused surgery.

In seventeen of the twenty-seven failures, repair was carried out. Nine of these revisions failed and eight were successful. In four of the nine failures, the patients were

Chart IX



relatively asymptomatic and were only slightly incapacitated. Two were moderately handicapped by pain, but used no support. One uses crutches, but refused surgery; another leads a sedentary existence; and one is an invalid.

In the seventy-seven cases in which some type of bone graft was used (Chart VII), the pseudarthrosis rate was 22 per cent. Nine of the failures occurred between the graft and the femur, four between the graft and the ilium. There were two instances of fracture of the graft and one of absorption of the graft, associated with infection; one failure occurred at the osteotomy site in the Brittain arthrodesis.

Chart VIII shows the postoperative knee pain present after removal of the plaster and completion of reconditioning. Of the patients in whom solid hip fusion developed, 9 per cent. of those in both the osteo-arthritis group and those with congenital dislocation under twenty-one years of age complained of knee pain and instability. In the poliomyelitis group, 22 per cent. had the same complaints.

Chart IX shows the presence of preoperative and postoperative back pain. In 31 per cent. of all fusions, the patients had some degree of backache, either after prolonged sitting in low chairs or associated with exertion. This usually subsided with rest. Several patients complained of back pain when lying in bed, unless a pillow was placed under the knee on the involved side.

Two patients over thirty-five with congenital dislocation of the hip complained of back pain before operation, but not afterward. In these instances the hip was fused in less than the average amount of flexion.

In 6 per cent. of the patients with osteo-arthritis, changes in the lumbar spine were noted in the roentgenograms before operation, but no complaints could be elicited. These patients had back pain after operation.

In eight patients, osteotomies were performed because of unsatisfactory position following fusion. In five of these union was satisfactory; non-union developed in one; two showed delayed union after one and one-half years, but had union at the end of two years.

There were seven postoperative infections, or an infection rate of 6 per cent. In three, the infection caused no significant delay in fusion. In two, union did not develop until two years after sequestration of the graft; two cases were complete failures. Of the cases where the outcome was altered by infection, two patients had osteo-arthritis; one, in the group over thirty-five years of age, had congenital dislocation; and one had had suppurative arthritis.

Four patients fell and sustained subtrochanteric fractures following solid hip fusion, and one had a fracture of the lower third of the femur. All of these fractures healed and the end results were satisfactory.

SUMMARY AND OBSERVATIONS

1. An analysis was made of 117 patients in whom hip-joint arthrodesis was performed in one Clinic.

2. The pseudarthrosis rate for the entire group was 23 per cent. and the mortality rate was 3.4 per cent. All deaths occurred in the osteo-arthritis group.

3. The rate of infection was 6 per cent.

4. Not all pseudarthroses are incapacitating. Of the fifteen cases in this series in which pseudarthroses ultimately developed, eight patients were relatively asymptomatic.

5. Back pain was present in all age groups following arthrodesis.

6. The gait was deliberate and acceptable in patients with neutral abduction-adduction without shortening.

7. The most normal gait resulted in patients with very slight adduction and no shortening.

8. The poorest gait was in patients with equal limb lengths and abduction; this created a relative lengthening of the fused side, not compensated for by the opposite extremity.

9. It appeared that the most certain method of producing arthrodesis was (a) intra-articular fusion, plus a bone graft, plus a nail of the Smith-Petersen type for internal fixation; or (b) intra-articular fusion plus the insertion of three Vitallium nails.

10. The first attempt to repair pseudarthroses of the hip resulted in 50 per cent. of failures.

11. It has definitely been demonstrated that bony fusion may occur after two years without further surgery.

12. The patients in this study were decidedly benefited by hip arthrodesis.

DISCUSSION

DR. M. N. SMITH-PETERSEN, BOSTON, MASSACHUSETTS: Dr. Stinchfield has presented an honest, unprejudiced analysis of the results of other surgeons. It is my function not only to congratulate him, but also

to clarify, criticize, and disagree with some of his conclusions. He has stated that the mortality rate was 3.4 per cent., the operative infections 6 per cent., and the pseudarthrosis rate 23 per cent. These are all high figures, but many of these operations were performed by the house staff; and we must also keep in mind that this study goes back to 1935, when the operative technique was not what it is today.

Seventeen of the twenty-seven pseudarthroses were revised; nine of these failed, eight were successful. I cannot resist the temptation to point out the difference in the attitude of the surgeon toward failure of arthrodesis and toward failure of mold arthroplasty. If an arthrodesis fails, it is due to some error in technique or to some unfavorable condition on the part of the patient, so the operation is revised. If a mold arthroplasty fails to produce a symptom-free hip, then the mold is to blame, so it must come out and an arthrodesis or an osteotomy must be performed. It would be very instructive if failures of mold arthroplasty were analyzed, the same as failures of arthrodesis, as to the cause of the failure, and if the arthroplasty were revised accordingly, rather than blaming the failure on the mold, discarding it, and then performing some other operative procedure.

The conclusion was drawn that, in congenital dislocation of the hip, arthrodesis was more successful before the age of twenty-one than after the age of thirty-five. The reason for this was not made clear in the paper, because the authors failed to point out that in the younger age group there were no bilateral dislocations; all eleven cases were unilateral. In the older age group, however, five of the nine cases were bilateral. Arthrodesis in bilateral congenital dislocation of the hip, or in any bilateral condition, is rarely, if ever, indicated. This, then, is the probable explanation for the high percentage of failures in the older age group.

Thirty-one per cent. of all patients having fusion complained of backache. This is a surprisingly low figure. A stiff hip must be compensated for by excessive function on the part of the spine, and such abnormal wear and tear sooner or later produce back symptoms.

DR. RUDOLPH S. REICH, CLEVELAND, OHIO: In discussing this excellent paper of Dr. Stinchfield and Dr. Cavallaro, little need be said about the 77 per cent. of patients who obtained solid bony union following hip-joint arthrodesis. The very fact that many of these patients participated in active athletic exercises after hip arthrodesis is adequate proof of good results.

As to the 23 per cent. of failures, it seems to me that this ratio is consistent with some series of cases that have previously been reported. As we all know, the method of obtaining bony fusion by means of internal fixation with the Smith-Petersen nail, or other types of nails or pins, without hip-joint arthrodesis or bone-grafting, has been popular recently. Our experience has been similar to Dr. Stinchfield's,—namely, that internal fixation alone is inadequate, but it is of great help when combined with hip fusion, either alone or in conjunction with a bone graft. I should like to ask the essayists their conclusions as a result of their use of trochanteric and iliac grafts in iliofemoral fusion,—I assume they call this the Hibbs fusion. We obtained more failures with the trochanteric graft than with the iliac graft, and we have abandoned the trochanteric graft. Of course, I am discussing the type of case in which that graft was done, and not the interarticular fusion.

The problem that has been perplexing us has been the recurrence of deformities in children following hip fusion. These patients have had adequate immobilization for approximately six months, were protected from weight-bearing thereafter, and have seemingly achieved good bony union, both clinically and roentgenographically. Still flexion-abduction deformities develop, and osteotomies have been required, as in eight cases of the authors' series. I should like to hear their opinion as to the cause. Is it the result of failure in obtaining solid bony fusion, which is more apt to occur in adults, or is it the result of continued bone growth around the solidly fused hip joint in a child?

This is a comprehensive report of a large series, and should be of great aid to us in evaluating the methods of hip fusion.

DR. STINCHFIELD (closing): This study was started three years ago, at which time it was felt that it might be wise to study all the arthroplasties and all the arthrodeses performed during the same period of time for identically the same pathological conditions, operated upon by the same surgeons, in the same institution. In June 1948 an end-result study of the arthroplasties of the hip was presented at the meeting of the American Medical Association in Chicago [1]. The end-result study of arthrodesis of the hip is now presented. A third paper, comparing the two methods, will be forthcoming.

The same percentage of pseudarthroses occurred with the trochanteric graft as with the iliac graft. In all cases where a trochanteric graft was used, an intra-articular fusion was also done. This series did not include arthrodesis of the hip for tuberculosis.

In this series of hip arthrodeses, five individuals were under the age of fifteen. Flexion deformity occurs more frequently in children. Our only explanation for this would be: (1) When a child is put in plaster, the atrophy of the extensors is more extensive than that of the flexors, which would tend to produce a flexion deformity. (2) The epiphyseal plate in a child is not closed, which gives a greater tendency for the limb to go into flexion than in an adult. (3) Children are much more apt to try to sit up and move around after they are put in plaster than are adults, which again predisposes to the flexion position.

1. STINCHFIELD, F. E., and CARROLL, R. E.: Vitallium-Cup Arthroplasty of the Hip Joint. An End-Result Study. J. Bone and Joint Surg., 31-A: 628–638, July 1949.

FRACTURES OF THE CALCANEUS

BY GEORGE E. WILSON, F.A.C.S., F.R.C.S., TORONTO, ONTARIO, CANADA

The calcaneus is clinically the most important bone of the foot, not only because of the frequency of its fracture, but also because of the severity of the resulting disability. This bone articulates with the cuboid anteriorly and the talus above. Inasmuch as the talocalcaneal articulation is subject to weight-bearing, it is the one to which attention will be chiefly directed, since disturbances in the relations of this joint are the cause of most of the disability in fractures of the calcaneus.

The superior aspect of the calcaneus may be considered as having three surfaces, an anterior horizontal surface, a middle oblique surface, and a large posterior surface, again horizontal but placed on a higher level (Fig. 1). Most of the anterior surface is non-articular, but a small area posterior to the anteromedial angle supports the anterior facet. Immediately behind this a strong shelf of bone, the sustentaculum tali, projects medially; triangular in outline, it is strongest and widest posteriorly and supports the middle facet. Together, these facets form a narrow concave oval, directed forward and somewhat laterally; frequently they are joined. The middle and anterior surfaces are separated from each other by a deep gutter, the sinus tarsi, into which is inserted the powerful interosseous talocalcaneal ligament. This surface is wholly occupied by the posterior facet, a wide oval with the long axis running downward and forward at an angle of approximately 45 degrees. It is convex in the anteroposterior direction and, about halfway down, the lateral half bends downward considerably more than the medial half. Since the outline of this facet is of great importance from the roentgenographic standpoint, we have considered this facet as having a lateral and a medial half. The posterior surface is non-articular; its junction with the middle surface marks the highest portion of the bone and is named the *posterior peak*. For purposes of description, we have named a line joining the anterosuperior with the posterosuperior angle of the bone the *ceiling line*.

The calcaneus is peculiar in that knowledge of the anatomy of the bone does not help greatly in interpreting the appearance of the roentgenograms; and, since a study of the films is the chief means of determining the changes in structure resulting from the fracture, it is of considerable importance to be able to interpret what one sees.

In order to familiarize himself with the significance of the various markings on the films, the author made a study of roentgenograms in conjunction with the normal dried bone, first placing identifying wires on the various points of interest. Lateral and plantar exposures were then made of the bone. In the former, the plate was placed on the lateral aspect of the bone, while in the latter it was placed vertically against the back of the bone and the rays were directed from the plantar surface, the ankle being at a right angle. In the lateral views marked differences were found in the appearance of the films with varying degrees of eversion and inversion of the foot. In full eversion (Fig. 1) the calcaneus resembles a Dutch sabot; anterior and posterior horizontal lines, representing the anterior and posterior surfaces, are joined by a convex oblique line, running downward and anteriorly at an angle of about 45 degrees. This oblique line corresponds to the lateral half of the posterior facet, the medial half being obscured by overlying bone. In this view neither the anterior nor the middle facet is seen, being likewise obscured by overlying bone. However, the sustentaculum tali, which supports the middle facet, can always be detected as a reniform area of density a little below the posterior aspect of the anterior surface. When the ceiling line is constructed with the foot in full eversion only one projection, the posterior peak, appears above it. In this view there is always a considerable amount of overlapping between the anterosuperior angle of the calcaneus and the head of the talus, as well as between the postero-inferior angle of the talus and the posterior peak of the calcaneus.

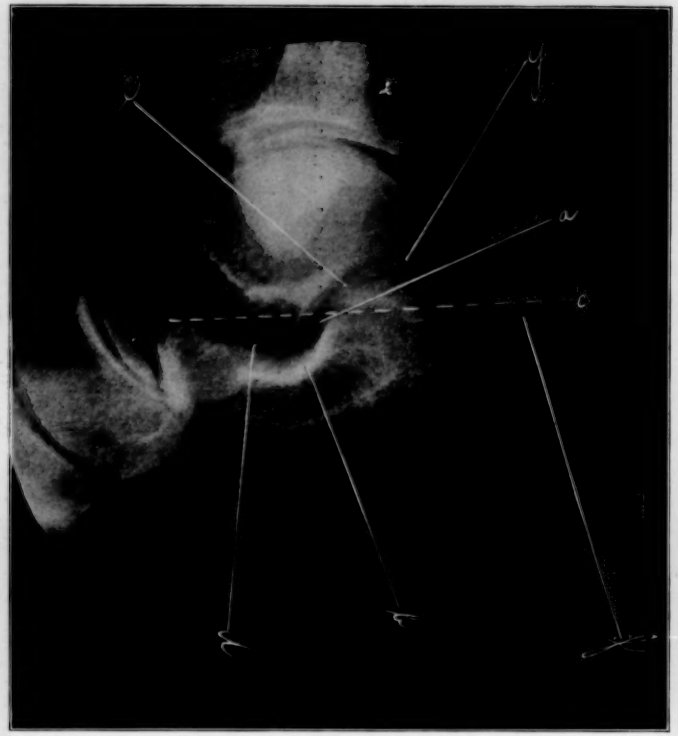


FIG. 1

Calcaneus with the foot in full eversion.

a: Lower part of posterior facet, lateral portion only showing.
b: Upper surface of sustentaculum tali, which is represented by the reniform outline below; anterior and middle facets cannot be seen.
c: Ceiling line. *d:* Anterior surface. *e:* Middle surface.
f: Posterior surface. *g:* Posterior peak.

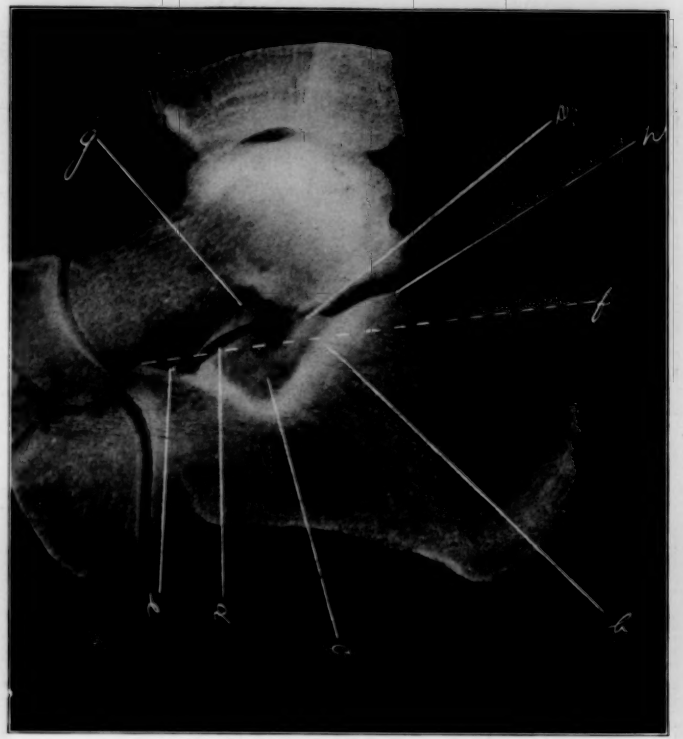


FIG. 2

Foot in full inversion.

a: Medial portion of posterior facet. *b:* Lateral portion of posterior facet.
c: Sustentaculum tali. *d:* Middle facet. *e:* Anterior facet.
f: Ceiling line. *g:* Anterior peak. *h:* Posterior peak.

FIG. 3

FIG. 4

Fig. 3: Plantar view of foot.
a: Sustentaculum tali. *b:* Tip of medial malleolus. *c:* Middle facet. *d:* Tip of lateral malleolus. *e:* Posterior facet. *f:* Notch on the posterolateral corner.

Fig. 4: A common variety of fracture, with the whole central portion depressed below the ceiling line. (Figs. 13-A and 13-B show other views of this same fracture.)
a: Os trigonum. *b:* Middle facet. *c:* Fissure running into calcaneocuboid joint. *d:* Posterior facet.

FIG. 5-A

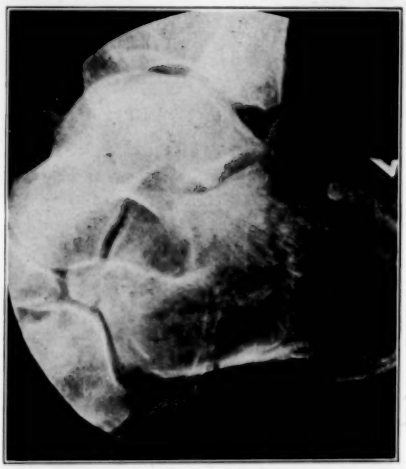


FIG. 5-B

Fig. 5-A: Split posterior facet.
a: Lateral portion of posterior facet is intact. *b:* Medial portion is considerably depressed. *c:* Apex of sustentaculum tali is not greatly disturbed.
Fig. 5-B: Showing reduction.

In full inversion (Fig. 2), a marked change in appearance takes place in the superior surface; this is due chiefly to the presence of the sustentaculum tali, which appears as a domelike mass, arising within the angle formed by the anterior and middle surfaces. Since its apex rises above the ceiling line, we have for convenience called it the *anterior peak*. The posterior portion of this projection is crossed by an obliquely directed shadow, produced by the medial half of the posterior facet, while the anterosuperior outline corresponds to the middle facet. In this view the lower portion of the lateral half of the posterior

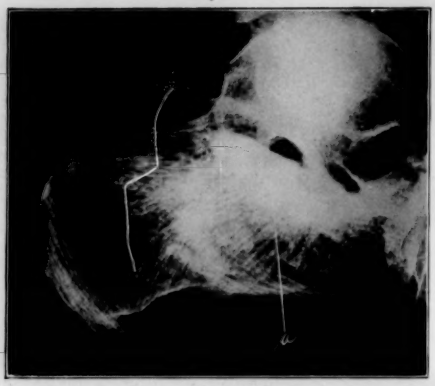


FIG. 6-A

Fig. 6-A: Split posterior facet.
a: Medial portion of posterior facet is markedly depressed.

Fig. 6-B: Plantar view of same fracture.
a: Lateral splitting. *b:* Medial splitting.

FIG. 6-B

FIG. 6-C

FIG. 6-D

Lateral and plantar views after reduction.

facet is not sharply defined, but it can usually be detected as a narrow band of dense bone, running downward and forward to the region of the sinus tarsi. Immediately anterior to the sustentaculum tali is the anterior facet, represented by a short horizontal line forming an obtuse angle with the middle facet. In full inversion, then, both the anterior and posterior peaks project above the ceiling line and overlapping is minimal, although the projection of bone in the anterior portion of the posterior facet of the talus may sometimes override the posterior aspect of the sustentaculum. In actual practice, it is impossible for the roentgenographic technician always to project the rays in the same direction, so that many intermediate views are common. As a matter of fact, very few of the films are taken with the foot in full inversion or eversion, so that in the majority some portion of the sustentaculum is visible. However, if one remembers that, as inversion proceeds, the sustentaculum gradually rises until its apex appears above the ceiling line, the interpretation should not be difficult.

In the normal plantar projection, because of the obliquity of the rays and other variables, there is necessarily a great deal of distortion, but with practice there should be no trouble in interpreting the films (Fig. 3). On the medial side and well forward, the sustentaculum tali is seen projecting medially, and about an inch farther forward the tip of the medial malleolus is evident; occasionally the middle facet can be seen immediately in front of the sustentaculum. On the lateral aspect the tip of the lateral malleolus can be seen opposite the sustentaculum, and the base of the fifth metatarsal makes its appearance half an inch forward and lateral to the malleolus. In a good film a line about an inch in length can be seen, starting from the lateral border and proceeding transversely toward the medial side. It is not quite so far forward as the sustentaculum tali and represents the important posterior facet. The medial surface is slightly concave; the lateral surface is somewhat convex and there is usually a small notch on the posterolateral corner. Obviously, if the rays are altered and if there is marked swelling, many of these features will be difficult or impossible to recognize.

Although there are numerous varieties of injury to this bone, some well-recognized types

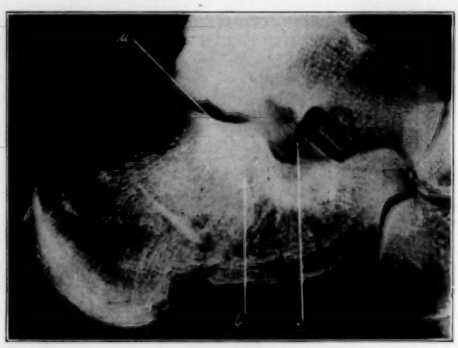


FIG. 7-A

Fig. 7-A: Split posterior facet.
a: Medial portion of posterior facet.
b: Lateral portion of posterior facet is depressed and rotated.
c: Portion of sustentaculum tali.

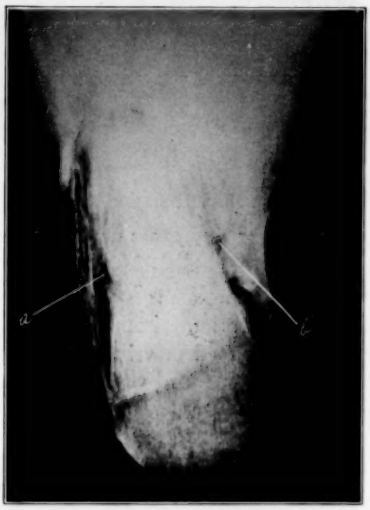


FIG. 7-B

Fig. 7-B: Plantar view of same fracture.
a: Lateral splitting. *b:* Medial splitting.

FIG. 7-C

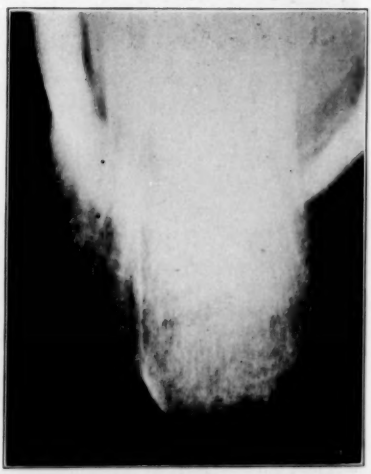


FIG. 7-D

Lateral and plantar views after reduction.

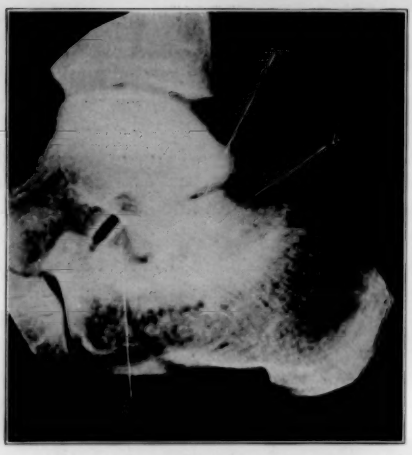


FIG. 8-A

Split posterior facet.
a: Medial portion is intact.
b: Lateral portion is considerably depressed and rotated.
c: Portion of sustentaculum tali.

FIG. 8-B

Lateral view after reduction.
a: Medial portion of posterior facet.
b: Upper margin of lateral half of posterior facet.

are seen in the roentgenograms. When these are examined from the side, the most common appearance is a depression of the area represented by the middle and posterior facets and the bone immediately adjoining them (Fig. 4). The fracture line, which is roughly Y-shaped or triradiate, commences just behind the posterior peak, and, after proceeding downward and forward for an inch or so, bifurcates. One limb proceeds anteriorly and upward into the region of the middle facet, while the other opens on the inferior surface. Frequently the lower limb is double or even treble, and each of these branches may terminate in a gap of about one-eighth of an inch on the inferior surface. In some instances a crack opens on the posterior surface, leaving a similar V-shaped gap

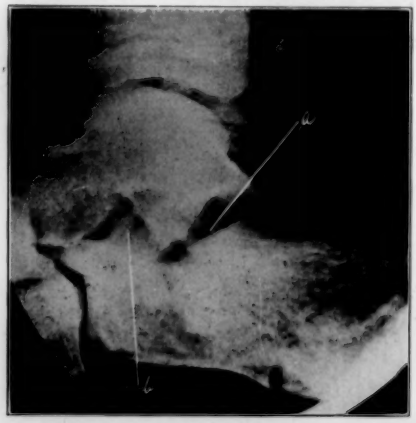


FIG. 9-A

Marked depression of posterior facet, sustentaculum tali remaining in position.
a: Posterior facet. *b:* Sustentaculum tali.

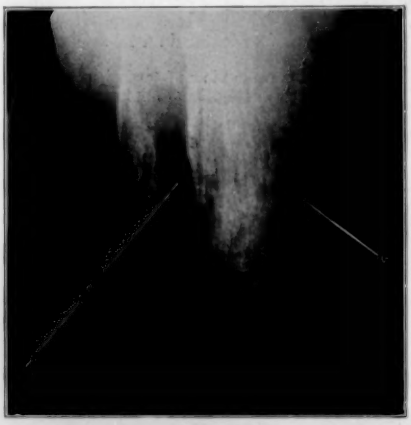


FIG. 9-B

Plantar view of the same fracture.
a: Wide lateral split.
b: Medial split. No reduction was attempted because of other severe injuries.

there. In the most severe cases, the superior surface of the bone is markedly concave, both peaks being below the ceiling line.

In almost every instance of this type, the normal relationship between the facets is disturbed, one being depressed more than the other. This is particularly true of the posterior facet, one half of which may be considerably depressed while the other half is but slightly distorted (Figs. 5-A to 6-D). If the lateral half is badly depressed, it is often rotated on a transverse axis so that the articular surface assumes a more vertical position (Figs. 7-A to 8-B). In many instances the sustentaculum does not follow the adjacent bone in its downward displacement, but remains in position (Figs. 9-A and 9-B), due possibly to the support given by the tendon of the flexor hallucis longus. In another fairly common

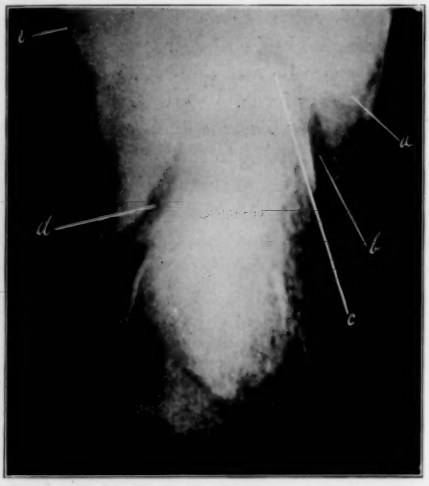


FIG. 10-A

FIG. 10-B

Fig. 10-A: Plantar view. Several fissures are present, but very little deformation of the bone.

a: Tip of lateral malleolus. *b:* Fissure going into posterior facet. *c:* Posterior facet.

d: Another fissure opening into posterior facet. *e:* Sustentaculum tali.

Fig. 10-B: Lateral view.

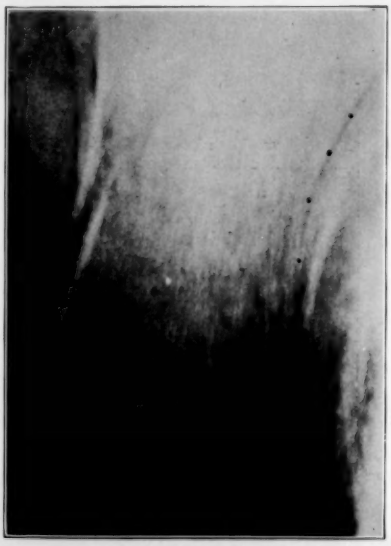


FIG. 10-C

Plantar view after reduction.

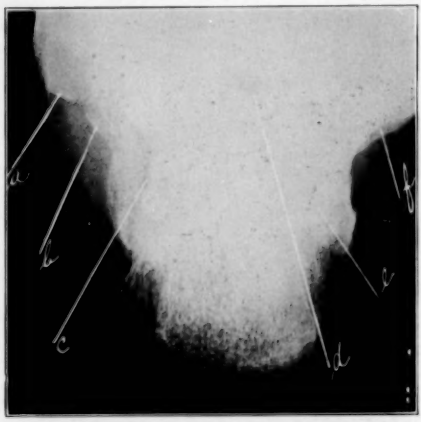


FIG. 11

Fig. 11: Distortion with apparent shortening.

a: Tip of fifth metatarsal. *b:* Lateral malleolus. *c:* Fissure going into posterior facet.

d: Posterior facet. *e:* Fissure going into posterior facet. *f:* Sustentaculum tali.

type, the fissure, while roughly Y-shaped, does not enter the talocalcaneal joint, at least in the lateral view; the compressed area descends *en masse* and only to a moderate degree. This probably represents the initial stage of the severe types just described.

Although in the lateral roentgenogram little or no deformation of the articular surface may be made out, it is unwise to conclude that the injury is a minor one, since the plantar view may show marked fissuring with involvement of the posterior facet (Figs. 10-A, 10-B, and 10-C). In another less common type, one or two fissures commence in the region of the middle facet and proceed in a more or less vertical direction toward the inferior surface. When double, these lines may either converge or diverge in their downward course. In a few instances, a small triangular fragment, about one-quarter of an inch to the side and slightly detached, can be seen at the anterosuperior border, or even the antero-inferior corner; while at other times a crack, which does not involve the articular surface, can be made out in the posterior third of the bone, either in the horizontal or vertical plane. In compression fractures of any severity, seldom will there be any deformation of the anterior facet, since it is outside the line of the compressing force.

When examined from the plantar aspect, the most obvious roentgenographic finding in compression fractures is widening of the bone, due to splitting in the vertical plane. The usual finding is a fissure on each side, starting an inch or so from the posterior surface and proceeding forward toward the calcaneocuboid joint (Figs. 12-A and 12-B). A wedge-shaped slab of bone is thereby split off from each side, the amount of separation depending upon the severity of the force. Next in frequency are the single splits; these are found most often on the medial side. Occasionally there are three or even four fissures. Obviously all these vertical fissures enter the subtalar joint, and many of them enter the calcaneocuboid joint as well. To visualize a fissure entering the calcaneocuboid joint, it is usually necessary to have an anteroposterior roentgenogram of the ankle joint, in which even the bulging and splitting on the lateral aspect of the calcaneus can frequently be made out as well. On account of the swelling, the difficulty of getting the foot at a right angle when taking

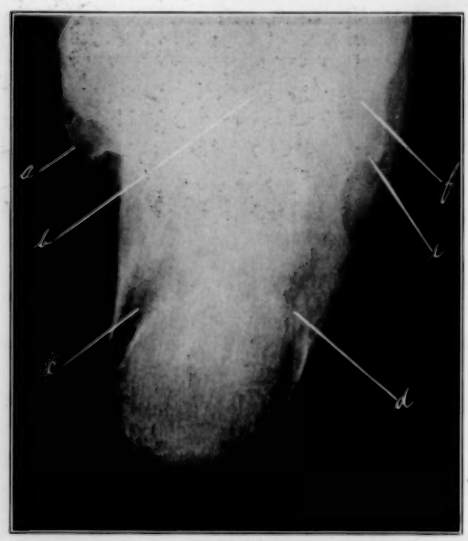


FIG. 12-A

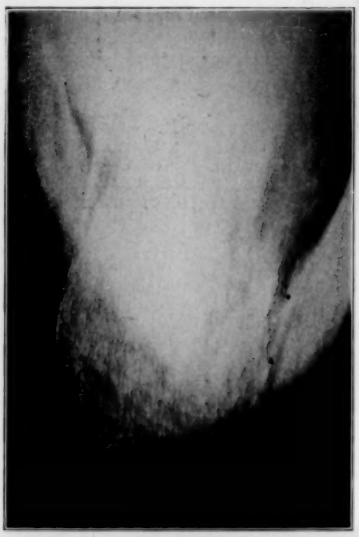


FIG. 12-B

Fig. 12-A: Plantar view of a double split.
a: Sustentaculum tali. *b:* Posterior facet. *c:* Medial split. *d:* Lateral split. *e:* Lateral malleolus. *f:* Fifth metatarsal.

Fig. 12-B: Plantar view after reduction.

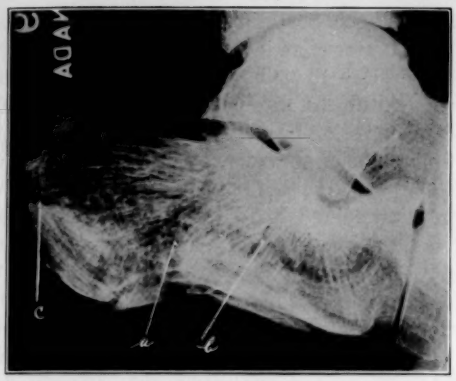


FIG. 13-A

Fig. 13-A: Marked compression fracture with three splits, two on the inferior surface and one on the posterior surface. Actual lengthening of one-quarter of an inch occurred.
a and *b:* Inferior splits. *c:* Posterior split.

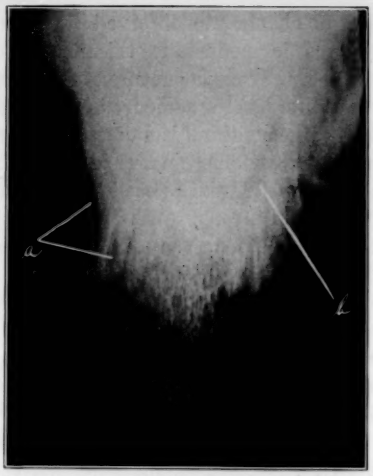


FIG. 13-B

Fig. 13-B: Plantar view showing double split and apparent shortening.
a: Medial split. *b:* Lateral split.

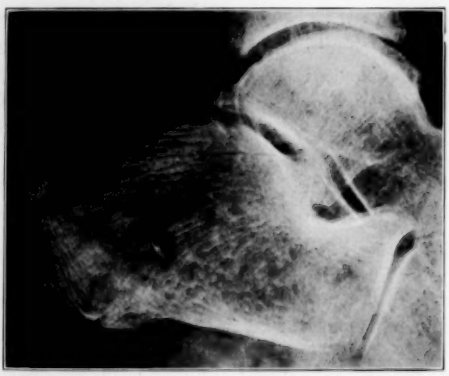


FIG. 13-C

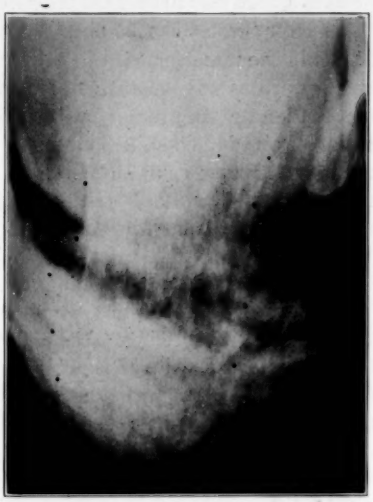


FIG. 13-D

Lateral and plantar views after reduction.

the roentgenogram, and the possibility of faulty technique, there is often considerable distortion (Fig. 11). In such instances the tip of the medial malleolus may overlap the sustentaculum, while on the lateral aspect the projections of the malleolus and the fifth metatarsal may almost coincide. In extreme cases of widening, these may be seen within the outline of the calcaneus. Frequently one or more fissures may be seen entering the posterior facet.

Some writers contend that the calcaneus is frequently shortened in cases of compression, but this statement is open to question. At any rate, no example of this has been found in the series studied by the author, and in the great majority of instances comparisons have been made with the opposite calcaneus by caliper measurement under fluroscopic vision prior to the setting of the fracture. True, in the plantar view the bone often appears shortened, but this is due to distortion, as already pointed out. In the vast majority of

cases no change was observed in the length of the bone. Sometimes, however, lengthening occurred; in one instance this amounted to one-quarter of an inch (Figs. 13-A, 13-B, 13-C, and 13-D). From the nature of the injury, it is difficult to see how shortening could occur. The author has the same feeling about elevation of the posterior portion of the bone, which some surgeons think is a common feature in compression fractures. In many respects the calcaneus resembles an arched bridge, the supports being the calcaneocuboid joint in front and the postero-inferior border behind. When the arch is driven downward by force transmitted through the talus, the posterior pillar, being less fixed, moves backward; the amount is influenced by the size of the gaps on the inferior surface and the extent of the widening.

On examining the injured foot, the most characteristic feature is the widening of the heel, which is very tender to pressure. Hemorrhage into the adjacent tissues is constant, and in severe cases the skin is tense and numerous ecchymotic areas can be seen. The diagnosis is not difficult, but fracture of the ankle as well as fracture-dislocation of the talus must be differentiated, as either of these conditions may complicate a compression fracture of the calcaneus. However, the location of the maximum swelling and tenderness over the calcaneus should point to a correct diagnosis. In any event, the final opinion must be based upon a study of the roentgenograms.

It is a good rule in surgical practice never to undertake a complicated procedure when a satisfactory result can be obtained by simple means. This principle is particularly applicable in the management of a compressed fracture of the calcaneus. The author, therefore, deprecates any operative attempt to replace the fragments; in view of the great number that may be present, such an attempt must at best be very incomplete. The introduction of pins is also objectionable, since they are apt to produce a distressing osteomyelitis, very difficult to cure. The object of treatment should be to restore, as far as possible, the facets to their normal position, and this can be accomplished best by closed methods. Before treatment is instituted, good lateral and plantar roentgenograms must be obtained and, preferably, an anteroposterior view of the ankle as well. These should always be taken with the same technique, in order that accurate comparisons may be made.

Having studied the films, we proceed with the setting. The method used, a modification of Cotton's procedure[1], has been employed at St. Michael's Hospital, Toronto, for several years, with quite satisfactory results. It is a combination of simultaneous traction, pounding, and lateral pressure, and is carried out with the aid of fluoroscopic vision, both in the lateral and plantar views. The patient having been given a spinal anaesthetic, a lateral view is obtained by placing the lateral aspect of the foot flat against the table and examining it in both eversion and inversion. Inversion is the more important, however, since by that means only can the position of the sustentaculum be determined. In obtaining a plantar view, the patient is turned onto his face and the feet are pulled down until they protrude beyond the end of the fluoroscopic table. A portable machine is then placed so that the rays are directed from below and pass obliquely through the sole, while the screen is held against the back of the heel. In this way, an excellent view can be had of the sides of the calcaneus.

A small, firm sandbag, which can be fitted snugly against the medial surface of the calcaneus, is laid on a solid portion of the table and the patient is turned on his side sufficiently to allow the medial side of the heel to be placed flat against the sandbag. This necessitates bending the patient's knee. A piece of heavy broom handle about eight inches long, to which a thick pad of felt has been nailed, is then placed upon the lateral aspect of the calcaneus, immediately below the lateral malleolus. While one assistant gets ready to pull up on the leg and another to exert countertraction with one hand on the dorsum of the foot and with the other on the back of the heel, the surgeon—having previously instructed the assistants to exert a strong simultaneous pull at the moment of impact—delivers a heavy blow on the broomstick with a four-pound mallet. The blow disimpacts

the fragments. As the talus moves upward, it pulls the depressed segment of the calcaneus with it, owing largely to the strong interosseous ligament. If disimpaction has been accomplished, crepitus can usually be made out; if the fragments have been elevated, the widening of the heel will have disappeared except for that caused by the oedema of the soft parts. If firm pressure, exerted by grasping the heel just below the malleoli between the thumb and index finger, is continued for a few minutes, the oedema will largely disappear and a fairly accurate opinion of the width can be obtained. The bone, with the foot in full inversion, is then examined with the fluoroscope to see that the depressed areas have been elevated sufficiently. If the sustentaculum is still depressed, the lateral aspect of the foot should be laid against the sandbag and the broomstick should be placed against the medial margin of the sustentaculum, which can always be felt just below and a little anterior to the medial malleolus. It should be given a sharp blow in a lateral and upward direction. If reduction has been secured, both peaks should be well above the ceiling line with the foot in inversion, and in the plantar fluoroscopic view the width should be fairly well restored. If undue widening remains, the manoeuvre may be repeated or, provided crepitus is obtainable, lateral pressure by the hands may be tried. If that fails, pressure may be applied by means of a Thomas wrench. The wrench should be tightened gradually, the handle being wiggled from side to side at the same time.

When satisfactory reduction has been accomplished, one can proceed with fixation by means of plaster-of-Paris. The patient should be prone with the feet hanging free beyond the end of the table. This helps the operator to judge the normal position of the heel, since he can compare it with the opposite side. Under normal conditions, the heel turns in a few degrees and this must be recognized. It also must be remembered that in a fracture of the calcaneus, if the heel is displaced at all, it is usually to the medial side, and it is not uncommon to see instances in which this deformity has not been corrected. A snugly fitting portion of stockinette, long enough to extend to the knee, is pulled over the foot and a moderate amount of raw cotton is wound about the ankle. A thick piece of sponge rubber, two inches by one and one-half inches, is then placed immediately beneath each malleolus, and over this the plaster is applied. This extends from just below the knee to the heads of the metatarsals, and the foot is allowed to fall into moderate plantar flexion. Just as the plaster is hardening, firm pressure is exerted on the sponge rubber, making sure that the pressure is as close to the tips of the malleoli as possible. If the swelling is moderate, the pressure is applied by grasping the heel from behind between the thumb and index finger, the right hand being used for the left heel and the left hand for the right heel; the pressure is maintained until the plaster has set. If the swelling is marked, however, it is better to use both hands and to compress the heel laterally between the base of the palms. The pressure must never be sufficient to cause necrosis of the skin. Indeed, if the reduction has been complete, no great pressure is required to keep the alignment satisfactory.

Postreduction plates are made the next day. If the setting is satisfactory the cast is left on for a month, unless there has been a great deal of swelling, in which event it is wise to change the cast in about ten days. After removal of the cast, the patient is encouraged to move the foot in various directions many times a day. If there is much tendency to swelling, it is wise to apply a pressure bandage for a couple of weeks; if physical therapy is available, it should be recommended. The patient should not bear weight on the limb, however, for about eight weeks from the time of the setting, and then only with the help of crutches and when he is wearing a fairly stout shoe. The time at which the patient is able to return to work depends partly upon the severity of the compression, but to a greater extent upon the completeness of the reduction. In fractures of moderate degree and good reduction, the man will often be able to resume work in three months; in the very severe injuries, if the reduction is satisfactory, it may be six months before he can get back to his job, and even then he may have some residual swelling about the ankle.

The treatment of compression fractures of the calcaneus which have been poorly reduced, resulting in a painful foot, does not come within the scope of this article. However, one should never be in a hurry to recommend arthrodesis of the subtalar joint, as there is no guarantee that such a procedure will eliminate the disability. Nature is a wonderful healer if given sufficient time, and it is remarkable what a good result may sometimes ensue even in those cases with considerable deformation of the posterior facet. It is gratifying at times to see all discomfort disappear by the simple device of placing a lift on the medial side of the shoe. Because a certain amount of deformity is compatible with good function, some surgeons refuse to attempt any correction, but this view is surely untenable. An obviously deformed heel gives the individual an excellent excuse, if he is so inclined, to make the most of his injury. Moreover, the presence of deformity makes it very difficult for the examiner to say that there is no disability, even though he can find no evidence that the man is suffering pain. There is no legitimate excuse, therefore, to avoid correcting a deformed heel, the result of a compression fracture of the calcaneus.

SUMMARY

Compression fractures of the calcaneus are serious injuries, because of the frequent derangement of the upper articular facets. Because the interpretation of the broken fragments is difficult, a study was undertaken to determine the significance of the various lines seen in the lateral and plantar roentgenograms of the normal bone; with the knowledge acquired, recognition of the various forms of displacement in cases of fractures became easier. Inasmuch as the appearance of the bone differs greatly when the roentgenogram is taken with the foot in eversion or inversion, this fact had to be taken into consideration when assessing the findings. An imaginary line connecting the anterosuperior with the posterosuperior angle we have called the ceiling line. In eversion only one projection, the portion of the bone immediately behind the posterior facet, lies above this line; while in inversion the sustentaculum tali also rises above it. The fractures are reduced by a combination of simultaneous traction and compression. Reduction of the fracture is always done with the help of the fluoroscope, the plantar view being obtained by having the patient lie on his face with the feet projecting beyond the table. An excellent view can then be obtained by directing the rays from below. Fixation is by plaster for a month only. This is followed by exercises without weight-bearing for another month, after which weight-bearing, commencing with the use of crutches, is allowed.

1. Cotton, F. J.: Os Calcis Fracture. Ann. Surg., **64**: 480–486, 1916.

PROGNOSIS IN POLIOMYELITIS *

BY RAYMOND E. LENHARD, M.D., BALTIMORE, MARYLAND

A survey, similar to one previously reported for 1941 [2], was made of the 1944 poliomyelitis epidemic in Maryland. In summarizing the findings, an effort was made to correlate information gained from a statistical analysis of the cases, from the recovery of patients, and from the recovery of individual muscles, in order to present the prognosis for poliomyelitis.

STATISTICAL ANALYSIS OF CASES

The 1944 epidemic was the largest recorded in Maryland,—a total of 499 cases or an incidence of 24.9 per 100,000 population. There were 16 deaths or 3.2 per cent. In the United States for that same year the incidence of poliomyelitis was 13.6 per 100,000 population, and the death rate was 7.2 per cent.

There were 295 males and 204 females, or a ratio of three to two based on the 1940 census of the State (50.2 per cent. males, 49.8 per cent. females). Only 6.8 per cent. were

TABLE I

AGE DISTRIBUTION AND INCIDENCE

Age Group (*Years*)	Patients Number	Patients Per cent.	Population (From 1940 census)	Incidence (Per 100,000)
Under 4	107	21.4	136,665	78.29
5 to 9	163	32.7	140,830	115.74
10 to 14	108	21.6	156,352	69.07
15 to 19	64	12.8	167,777	38.14
20 and over	57	11.5	1,219,620	4.68
Total	499	100.0		

TABLE II

DEATHS FROM EACH TYPE OF POLIOMYELITIS

Type	Patients Number	Patients Per cent.	Deaths Number	Deaths Per cent.
Bulbar	47	9.4	13	81.2
Bulbospinal	62	12.4	3	18.8
Spinal	390	78.2	0	0
Total	499	100.0	16	100.0

negro patients, whereas the colored population of Maryland is 16 per cent. of the total. There were 181 cases or 36.3 per cent. in Baltimore, which has 50 per cent. of the total population of the State. The other 318 cases were in the twenty-three counties of Maryland. Four of these counties have large towns, are more densely populated than the others, and comprise 25 per cent. of the county census. In these four counties 236 cases occurred, or 47.3 per cent. of the total epidemic.

The 1944 epidemic of poliomyelitis in Maryland had a higher attack rate than that

* The survey was made possible by a grant from the National Foundation for Infantile Paralysis, Inc.

TABLE III

PERIOD OF HOSPITALIZATION FOR 384 PATIENTS *

Hospitalization	No. of Patients	No. of Hospital Days		Average Days per Patient
		Isolation	Convalescent	
During isolation period only	140	2,310		16.5
During both isolation and convalescent periods	213	4,986	30,798	168
During convalescent period only	31		4,039	130
Total				
During isolation period	353	7,296		20.5
During convalescent period	244		34,837	143

* 115 patients were not admitted to the hospital.

TABLE IV

ANALYSIS OF CASES IN WHICH RESPIRATOR WAS REQUIRED

Age Group	Sex		Race		No. of Extremities Involved	No. of Days in Respirator	Hospital Days			Category †							
	M.	F.	W.	C.			Isolation	Convalescent	Total	1	2	3	4	5	6	Died	Not Examined
Under 4	2		2		4	46	153	300	453	1						1	
5 to 9	3	2	5		6	21	46	689	735					1		3	1
10 to 14	6		6		16	395	500	1,026 *	1,526	1		1			2	1	1
15 to 19	4	1	4	1	14	866 **	668	715 *	1,383						3	2	
20 and over	1	5	6		20	480	525	628	1,153				2	1	1	2	
Total	16	8	23	1	60	1,808	1,892	3,358	5,250	2	0	1	2	2	6	9	2

* One child is still in hospital at time of survey (November 15, 1946).

** One child is still in respirator at time of survey (November 15, 1946).

† This refers to a grouping of cases in terms of recovery, as explained under "Recovery of Patients".

TABLE V

END-RESULT COMPARISON OF TWO EPIDEMICS

Classification	1941 Epidemic		1944 Epidemic	
	No. of Patients	Per cent.	No. of Patients	Per cent.
Good recoveries				
No paralysis	52	18	51	13
Complete recovery	96	34	172	44
Subnormal	47	16	27	7
Slight residual	40	14	58	15
Poor recoveries				
Moderate residual	32	11	47	12
Marked residual	14	5	25	7
Complete residual	6	2	9	2
Total patients	287		389	
Good recoveries		82		79
Poor recoveries		18		21

in the country as a whole, but a lower death rate. The incidence was low in the negro population; it was higher in the males and higher in the densely populated areas.

An analysis of the age distribution of the incidence according to age groups is shown in Table I. Four patients were under one year and eleven patients were over thirty years of age. The youngest patient was two months old; the oldest, forty-one years. The attack rate was apparently twenty-five times greater in the age group from five to nine years than in the adult group.

The types of poliomyelitis in this epidemic and the deaths from each type are classified in Table II. There were no deaths in the spinal group. Three deaths occurred in the bulbospinal group: One patient died on the sixth day; one patient was in a respirator for thirty-six days and, while still in a hospital, died of pneumonia on the 447th day; one patient died of pneumonia on the 305th day, while still in a respirator. The thirteen deaths in the bulbar group occurred from one to eleven days after the onset of poliomyelitis:

Number of Patients	*Days after Onset*
1	1
2	2
5	3
2	4
1	7
1	9
1	11

One hundred and fifteen patients were not admitted to a hospital. There were 140 patients admitted to an isolation hospital for an average stay of sixteen and one-half days. Thirty-one patients were admitted for convalescent care only, and 213 patients who had been admitted to an isolation hospital were transferred to another hospital for convalescent care (Table III).

Twenty-four patients required the use of the respirator (Table IV). Seven of these cases were of the bulbar type: Six patients died on or before the ninth day; one survived after being in the respirator for eighteen days. Seventeen were of the bulbospinal type: One patient died in the respirator on the sixth day and one died on the 305th day in the respirator. Two were classified as chronic respirator cases: One required 328 days in the respirator; the other is still in a respirator after two and one-half years. Thirteen are classified as convalescent respirator cases and remained in the respirator for the following periods of time:

No. of Cases	*No. of Days*	*No. of Cases*	*No. of Days*
2	3	2	24
1	12	1	26
1	14	2	36
1	16	1	40
1	23	1	74

The average period spent in the respirator in this group was 24.4 days.

RECOVERY OF PATIENTS

For the two-year follow-up, 389 patients, or 78 per cent. of the total, were available for the survey. Most of those not available had moved from the State. The patients surveyed had a complete examination of muscles by Mr. H. O. Kendall, chief physical therapist of the Children's Hospital School, Baltimore. They were also examined by an orthopaedic surgeon for an estimation of functional disability or deformity. A tabulation and classification were done in order to show the amount of recovery or the gross involvement of the patients. A description of the categories is as follows:

1. *Normal* (223 patients). This included all patients who showed no muscle weakness, as determined by complete muscle tests. Fifty-one non-paralytic patients were included.

2. *Subnormal* (twenty-seven patients). This included the patients who had slight weakness in isolated muscles (usually about 70 per cent. of normal), but not sufficient weakness to cause a functional disability. There was no visible handicap.

3. *Slight Residual* (fifty-eight patients). This included chiefly those patients who had involvement of one foot or one shoulder, or moderate abdominal weakness. Any involvement from zero up to and including 60 per cent. strength in the muscles was included; zero, trace, or 10 per cent. was listed as zero, and 20 per cent. to 60 per cent. were referred to as "weak" in the following description of cases:

Weak right shoulder		1
Weak left shoulder		1
Left foot		20
Zero tibialis anterior	1	
Weak tibialis anterior and tibialis posterior	6	
Weak gastrocnemius	3	
Entire foot weak	9	
Weak peronei	1	
Right foot		14
Zero tibialis anterior	2	
Weak tibialis anterior and tibialis posterior	7	
Weak gastrocnemius	2	
Entire foot weak	3	
Moderate abdominal weakness (about 50 per cent. of normal)		6
Weak opponens and slight abdominal weakness		1
Weak opponens and weakness of anterior neck		1
Weak tibiales and glutaeus medius, left		3
Weak tibiales and glutaeus medius, right		3
Quadriceps 40 per cent. and tibiales 60 per cent.		1
Quadriceps 40 per cent.		1
Quadriceps 20 per cent. and medius 30 per cent.		1
Weak foot and slight abdominal weakness		1
Weak deltoid and anterior neck weakness		1
Tibialis anterior and hamstrings weak		1
Glutaeus medius 40 per cent.		1

4. *Moderate Residual* (forty-seven patients). This included all patients with involvement of one entire extremity or one portion of each of two extremities, as well as a few with mixed weakness of isolated muscles. The involvement might be complete paralysis or

TABLE VI

RECOVERY ACCORDING TO AGE AND SEX

Age Group (*Years*)	Normal and Good Recoveries (Categories 1, 2, and 3)	Poor Recoveries (Categories 4, 5, and 6)	Incidence	Good Recoveries within Age Group (*Per cent.*)
Under 4	68	13	Second	84
5 to 9	108	20	Highest	84
10 to 14	80	14	Third	85
15 to 19	33	20	Fourth	62
20 and over	19	14	Fifth	58
Total	308	81		
Males *	196	39		
Females	112	42		

* In this survey, more males were affected than females; but the percentage of good recoveries was higher among the males.

TABLE VII

TYPE OF INVOLVEMENT IN RELATION TO AGE GROUP AND END RESULT

Type	Age Group	Category								Total
		Complete Recovery	Sub-normal	Slight Residual	Moderate Residual	Marked Residual	Complete Residual	Died	No Examination	
Bulbar	Under 4....	4	0	0	0	0	0	1	1	6
	5 to 9.....	8	1	0	0	0	0	3	1	13
	10 to 14....	9	1	0	0	0	0	4	2	16
	15 to 19....	3	0	0	0	0	0	2	0	5
	20 and over.	3	0	0	0	0	0	3	1	7
	Total.....	27	2	0	0	0	0	13	5	47
Bulbo-spinal	Under 4....	1	0	0	0	0	0	1	2	4
	5 to 9.....	15	0	3	2	0	0	0	4	24
	10 to 14....	11	0	2	1	1	1	0	2	18
	15 to 19....	2	0	2	0	0	3	1	0	8
	20 and over.	0	0	0	3	2	1	1	1	8
	Total.....	29	0	7	6	3	5	3	9	62
Spinal	Under 4....	43	5	15	10	2	1	0	21	97
	5 to 9.....	56	6	19	9	9	0	0	27	126
	10 to 14....	43	5	9	7	3	1	0	6	74
	15 to 19....	19	5	2	11	4	2	0	8	51
	20 and over.	6	4	6	4	4	0	0	18	42
	Total.....	167	25	51	41	22	4	0	80	390
Grand Total.....		223	27	58	47	25	9	16	94	499

weakness up to and including 60 per cent. of muscles. Of the forty-seven extremities, five were flail or almost flail. They may be tabulated as follows:

Left arm	5
Right arm	2
Left leg	10
Right leg	11
Both feet	8
Both shoulders	2
Mixed weakness, approximately equivalent to the weakness of one extremity	9

5. *Marked Residual* (twenty-five patients). This included chiefly patients with involvement of two extremities, complete paralysis, or weakness up to and including 60 per cent. muscles (major muscles). In some cases a hip, a foot, or a knee was normal in strength. Eleven of the fifty extremities involved were zero and eight had muscles grading 30 per cent. or under, so that nineteen were grouped as flail or almost flail. They were classified as follows:

Bilateral lower extremity	23
Left arm and right leg	1
Bilateral upper extremity	1

6. *Complete Residual* (nine patients). All of these patients had involvement of all four extremities and trunk. Three patients had all four extremities flail or almost flail;

TABLE VIII

PERIOD AT WHICH MUSCLES ATTAINED MAXIMUM RECOVERY

Period of Maximum Recovery	1932–1938 Poliomyelitis Study		1944 Poliomyelitis Survey	
	No. of Muscles Attaining Maximum	Per cent.	No. of Muscles Attaining Maximum	Per cent.
5 years	3	0.4		
4 years	5	0.7		
3 years	7	1.0		
2 years	5	0.7	19	1.8
21 months	5	0.7		
18 months	26	3.7	59	5.7
15 months	38	5.4		
12 months	55	7.8	66	6.4
9 months	76	11.0	114	11.1
6 months	112	16.0	223	21.7
3 months	70	10.0	282	27.3
6 weeks	176	25.1	185	18.0
First examination	123	17.5	82	8.0
Total	701	100.0	1,030	100.0

one was a chronic respirator case. Of the thirty-six extremities, nineteen were flail or almost flail.

In 1941 a similar survey had been made. The end results of the 1941 and the 1944 epidemics are compared in Table V. The cases classified as subnormal showed almost total recovery; the weakness of muscles was graded against a high standard which, in the hands of other examiners, might be called normal. Those cases classified as "slight residual" represented a very limited extent of involvement, and in most instances stabilization would be possible by operation to the point of minimal functional handicap. The cases classified as normal, subnormal, and slight residual are grouped together as "good recoveries". The percentage of good recoveries was approximately the same in the two epidemics. Similarly, the categories of moderate to complete residual involvement of extremities were essentially the same in the two epidemics. The inference may be that the epidemics were not very dissimilar, if analyzed carefully.

An analysis of recovery with respect to age and sex is seen in Table VI. An analysis in relation to age group and type of poliomyelitis involvement (Table VII) shows that all of the patients with bulbar involvement either died or recovered completely. None of the adults with bulbospinal paralysis attained a good recovery.

RECOVERY OF MUSCLES

In addition to the analysis of the recovery of patients, the author rechecked some facts established in a previous survey in 1940, in regard to the recovery of muscles. In both the 1940 survey (covering statistics from 1932 to 1938) and the 1946 survey of the 1944 poliomyelitis cases, the muscles studied were the glutaeus maximus, glutaeus medius, inner hamstrings, outer hamstrings, quadriceps, gastrocnemius, and tibialis anterior in the lower extremity, and the middle portion of the deltoid, biceps, triceps, and opponens pollicis in the upper extremity. These eleven muscles were chosen as representing a fair group, both from the standpoint of importance and because they are subjected to strain in ordinary functional activity. In the 1940 survey, 701 muscles were studied from the records of seventy-four patients; and in the 1946 survey, 1,030 muscles, from the records of 123 patients at the Children's Hospital School. The period at which the muscles attained their

maximum recovery is seen in Table VIII. Although the 1946 survey is limited to a two-year follow-up, the findings seem to confirm those in the 1940 study.

At the first examination in the 1944 survey, 8 per cent. of the muscles had reached maximum recovery. At the end of three months, maximum recovery had been reached by a total of 53.3 per cent. of the muscles, and at six months by a total of 75 per cent. By the end of eighteen months, 98.2 per cent. had attained maximum recovery. This does not necessarily mean complete recovery to normal power, but that recovery had ceased at the time indicated on the chart. If treatment had stopped after the first three months, there is a possibility that 47 per cent. of the muscles might have lost the chance to attain maximum improvement. If treatment had ceased after six months, 25 per cent. of the muscles might have lost this opportunity.

However, one does not continue to treat muscles and hope for recovery which cannot be obtained. A maximum point of recovery is reached within approximately eighteen months after the onset of the disease; but previous studies have shown that, if no improvement in power is noted after a three-month period (provided treatment has been adequate and accurately done), the maximum probably has been reached, and there is nothing to be gained by further treatment of the patient in the convalescent phase. If muscles do not improve after three months of treatment, only 5.5 per cent. of them show improvement thereafter. If they do not improve after six months of treatment, only 2.4 per cent. can be expected to show any improvement by continued treatment. Only by careful observation of muscle power can we know what is being accomplished and how long to persist with treatment.

One might be interested to know what opportunity muscles have to recover from poliomyelitis, especially those that are initially weak. Table IX shows what results can be expected of muscles starting at various levels of initial power, with a comparison of the results obtained in 1932 to 1938. A muscle attaining 80 per cent. power is considered a good muscle,—able to complete the arc of motion against gravity and a medium amount of resistance several times without showing signs of fatigue. In the 1944 epidemic, 7.3 per cent. of muscles graded as zero made good recovery; 23.3 per cent. of the 10 per cent. muscles recovered; and 50.8 per cent. of the 20 per cent. muscles recovered. Muscles starting at higher levels recovered more readily.

TABLE IX

MUSCLES ATTAINING A FINAL GRADE OF 80 PER CENT. OR MORE

First Examination Grade (*Per cent.*)	1932 to 1938 Study			1944 to 1946 Study		
	No. of Muscles Graded	Muscles Attaining 80 Per cent. or More		No. of Muscles Graded	Muscles Attaining 80 Per cent. or More	
		No.	Per cent.		No.	Per cent.
0	200	13	6.5	273	20	7.3
10	58	24	41.4	60	14	23.3
20	86	43	50.0	59	30	50.8
30	82	67	81.7	52	37	71.1
40	57	46	80.7	64	49	76.5
50	41	36	87.8	54	48	88.8
60	64	57	89.0	291	282	96.9
70	56	54	96.4	111	109	98.2
80	57	57	100.0	66	66	100.0
Total	701	397	56.6	1,030	655	63.6

TREATMENT

Three hundred and twenty-five patients were admitted to hospitals for the isolation period. When treatment was required beyond the isolation period, the patients were admitted to other hospitals for convalescence.

At the Children's Hospital School, the treatment consisted of rest for the weak or paralyzed muscles in positions to relieve strain or stretch, and physical therapy in the form of heat, massage, and exercises specifically outlined for and applied to each patient, dependent upon information obtained after careful examination for muscle power. The treatment was increased in frequency and magnitude as the muscles demonstrated recovery.

The rationale of such treatment is based upon clinical observations controlled by examination of muscles, done by trained therapists whose accuracy has been clearly demonstrated. Mr. Kendall and his staff of physical therapists have had years of experience with hundreds of patients and hundreds of thousands of muscle examinations[1]. The author knows of no more accurate method of charting the course of muscle recovery.

In 1940 the records of cases of the preceding eight years were carefully reviewed and it was determined that, once the original pattern of muscle weakness has been established, there is a persistent recovery of power, until a maximum is reached. This does not apply to muscles graded zero or slightly better; here weakness remains because of damage to the central nervous system. There is no recession of power except under conditions of improper treatment. Careless handling and overtreatment may superimpose stretch weakness, weakness due to fatigue, or strain on joint structures to such an extent that maximum recovery may be delayed or never attained.

The application of heat is helpful in relieving soreness in muscles and in relaxing them, but should be prescribed in moderation. If used to excess, it may be harmful. In an analysis of the 1944 epidemic it was found that, of those patients receiving hot-pack therapy during the first few weeks of the disease, 73 per cent. made a good recovery; of those who did not receive hot-pack therapy during the first few weeks of the disease, 87 per cent. made a good recovery.

The extremities are not held in rigid fixation by solid casts, but plaster shells are used for the lower extremities and wire splints for the upper extremities to obtain positions of rest for the muscles. The joints are not allowed a complete range of motion during the phase when the muscles are weak or painful, but the range is increased as recovery is demonstrated. Massage and exercise are given with care to avoid fatigue, and the dosage is prescribed in accordance with the muscle power.

In the 1944 epidemic, as in previous years, it was evident that muscles involved by poliomyelitis responded to supportive treatment and carefully controlled physical therapy. Unless treatment was faulty, the improvement was continuous until a maximum recovery had been obtained. This was demonstrated over and over again. If, however, the muscles were fatigued by lack of support, if the patients were allowed to stand and walk too soon, if they were given curare or prostigmine, if the muscles were stretched and no heed given to supportive therapy, the muscles remained weak. Such muscles improved markedly after supportive care had been given, regardless of the time which had elapsed since the onset of the poliomyelitis. One can be reasonably sure that the muscles might have been much better had they been supported immediately after the onset of the disease.

Treatment for the poliomyelitis patient is commonly translated into an urge to do something,—to give heat in abundance, to give massage, exercise, and motion. The more that is done for the patient, the better the therapist feels. The greatest need in the early stage of convalescence is rest for the patient and for the muscles involved. They do not require, nor should they have, much physical therapy. The importance of vigorous treatment can be emphasized so much that the recovery of patients is actually retarded. This becomes apparent as soon as protective care is employed. How much harm is done to mus-

cles vigorously treated cannot be determined, but unless treatment is carefully managed, a superimposed weakness persists.

The posterior muscles of the trunk and legs are frequently tight. Some therapists accentuate the necessity for stretching them and insist upon extreme flexibility. Emphasis upon stretching back and hamstring muscles can result in a degree of flexibility far beyond normal and may result in an increased number of deformities, especially scoliosis, kyphosis, hyperextension of the knees, and lordosis. It is more desirable to have a back that is tight than one which collapses from muscle and ligament weakness.

Until such time as poliomyelitis can be prevented or attacked adequately in the acute stage, the problem remains of dealing with paralysis in the subacute stage. Apparently the recovery rate of epidemics is approximately 80 per cent., as demonstrated in two large epidemics in Maryland and as reported in other states. Approximately 20 per cent. of the patients have fairly extensive residual involvement of muscles. The excellence of any treatment can be measured by the amount of residuum in deformity and perhaps by the degree of weakness. The author believes that recovery can be aided by treatment and that muscles can lose power markedly by abuse or neglect. A lack of reserve power has been observed in muscles that have made an apparently complete recovery according to clinical tests, which makes it seem wise to avoid the fatigue of overtreatment or of exertion.

DEFORMITIES

The survey is a two-year follow-up study and, while it represents the probable limit of improvement of muscles, it does not relate the end of the story of deformities. However, it appeared worth while to survey the extent of deformities at the end of this two-year period. Wherever there is imbalanced power in antagonistic muscle groups, deformities can occur during the convalescent stage; and, unless supervision of the patient is maintained, they will occur later. This survey was the first opportunity in Maryland to compare two series of cases. There were 150 cases treated by non-supportive methods and 150 cases treated by supportive methods. In the former series there were thirty-five deformities in twenty-two cases; in the latter series, nineteen deformities in fifteen cases. Fewer deformities occurred in those cases treated by supportive therapy and careful physical therapy.

SUMMARY

A survey of the 1944 epidemic of poliomyelitis in Maryland, with data concerning the incidence, the mortality, and the morbidity in relation to age, sex, and the types of the disease, has been compared with a survey of the 1941 epidemic in Maryland. The results of the two epidemics were similar, except that slightly more patients had residual involvement in 1944.

Approximately 80 per cent. of the patients obtained a good recovery, and apparently treatment aids in attaining maximum recovery. Treatment should be carefully supervised and guided by muscle examinations to minimize the residual involvement in those who do not make a good recovery.

Supportive treatment is important. Delay in recovery and persistence of weakness have been observed in patients allowed to fatigue muscles by overtreatment, too much activity, or lack of necessary support. Treatment given without regard to the protection of muscles results in a greater degree of residual involvement and more deformities.

NOTE: The author is indebted to Mr. and Mrs. Henry O. Kendall for the examination and recording of muscle power, and to Mrs. Marta Rohrer for the compilation of data.

REFERENCES

1. KENDALL, H. O., and KENDALL, F. P.: Normal Flexibility According to Age Groups. J. Bone and Joint Surg., **30-A**: 690–694, July 1948.
2. LENHARD, R. E.: The Results of Poliomyelitis in Baltimore. J. Bone and Joint Surg., **25**: 132–141, Jan. 1943.

SURGICAL DIVISION OF THE PATELLAR RETINACULA TO IMPROVE EXTENSION OF THE KNEE JOINT IN CEREBRAL SPASTIC PARALYSIS

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The inability of patients with cerebral spastic paralysis to extend the knee joint completely, because of partial, persistent, and passively correctable flexion, has been demonstrated. The condition has been improved satisfactorily by the method of Chandler. In his operation, the hamstring muscles and the capsular restriction are released and the patellar ligament is advanced distally by transfer of the tibial tubercle, or the patellar tendon is shortened.

The inability of the quadriceps to completely extend the tibia at the knee joint presents an interesting problem. The quadriceps is enclosed by the fascia lata of the thigh and glides freely within it above the knee. This freedom is limited at the patellar level. The limitation is brought about by the arrangement of both the fascial and the muscle structures. Fibers of the fascia lata which extend to the borders of the patella are referred to by Cunningham as the patellar retinacula. Laterally, these fibers are continuous with the thickening of the fascia lata, known as the iliotibial band, which attaches directly to the lateral tibial condyle, anterior to the proximal fibular articulation. Distally, the fascia lata becomes continuous with the deep fascia and intermuscular septa, and is attached to the tibia and fibula and to the tendons as they cross the knee. Medially, the deep fascia is bound to the tendons of the sartorius, gracilis, and semitendinosus. Of particular importance, the fascia lata is fused with the tendinous slips of insertion for the vasti (Fig. 1). These muscles, in addition to inserting on the patella, send slips to the condyles of the tibia (Fig. 2). These slips are referred to by Morris as patellar retinacula.

Thus, the pull of the quadriceps as a whole is primarily on the tuberosity of the tibia

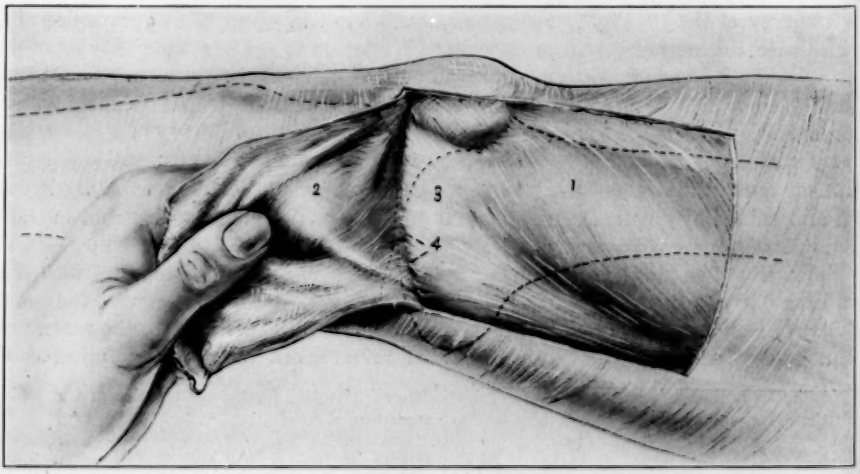

FIG. 1

Fusion of the fascial and muscular retinacula, medial side of knee joint.
1: Vastus medialis.
2: Fascial retinaculum.
3: Muscular retinaculum.
4: Fusion line of musculofascial retinacula.

by way of the patellar ligament, secondarily by way of the slips from the vasti to the tibial condyles on each side of the patellar ligament, and also, through fusion of these insertions with the fascia lata, to its attachments on the tibia and fibula. Thus, in extension of the knee joint, the pull is not only in the median line of the extremity through the patella, but also to the sides, through the fibrous and the muscular patellar retinacula (Figs. 3 and 4).

These anatomical facts are basically important in the flexed condition of the knee, found in cerebral spastic paralysis. The prolonged knee flexion causes stretching of the patellar ligament, but there is less stretching of the structures medial and lateral to the ligament. The amount of stretching is greatest in the arc of the patellar ligament, and decreases as the radial center of this is approached (Fig. 5).

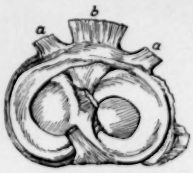

FIG. 2

Fig. 2: Insertion of patellar ligament and muscular retinacula into tibia.
a: Muscular retinacula.
b: Patellar ligament.
(Redrawn from Morris' *Human Anatomy*, Ed. 10, Fig. 346, p. 349.)

Fig. 3: Lateral aspect of knee joint. Retinaculum (*a*) is divided in the surgical procedure.
a: Muscular retinaculum.
b: Patellar ligament.
(Redrawn from Toldt: *Anatomischer Atlas für Studierende und Ärzte*, Fig. 469, p. 231. Berlin, Urban und Schwarzenberg, 1911.)

FIG. 3

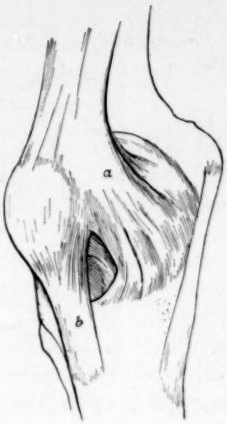

FIG. 4

Fig. 4: Medial side of knee joint. Retinaculum (*a*) is divided in the surgical procedure.
a: Muscular retinaculum. *b:* Patellar ligament.
(Redrawn from Gray's *Anatomy of the Human Body*, Ed. 24, Fig. 345, p. 334. Philadelphia, Lea and Febiger, 1944.)

In these spastic patients, the extensor action of the quadriceps is restricted by the fibrous and the muscular retinacula, attached to the sides of the patella and to the tibial condyles at points medial and lateral to the mid-line of the knee. This is due to the fact that these retinacula are more nearly normal in length than the patellar ligament, which has been stretched; thus the final extension pull is not exerted upon the tuberosity, but rather upon the medial and lateral aspects of the tibia, and complete extension cannot be produced.

SURGICAL TECHNIQUE

To accomplish the retinacular release, surgical division of these structures is performed. The hamstrings having been released, the patient is placed in the supine position with knees extended. An incision, three inches long, is made one-half inch lateral to the lateral patellar margin and parallel to it, beginning at the level of a point half-way between the distal end of the patella and the tibial tuberosity. The fascial retinaculum is displayed and divided parallel to the patellar ligament and the patella, and then the incision is curved posteriorly to a point anterior to the femoral attachment of the fibular collateral ligament. As this is done, the illiotibial band is also cut. The next structure seen is the muscular retinaculum (attachment of the vastus lateralis), and this is divided in the same

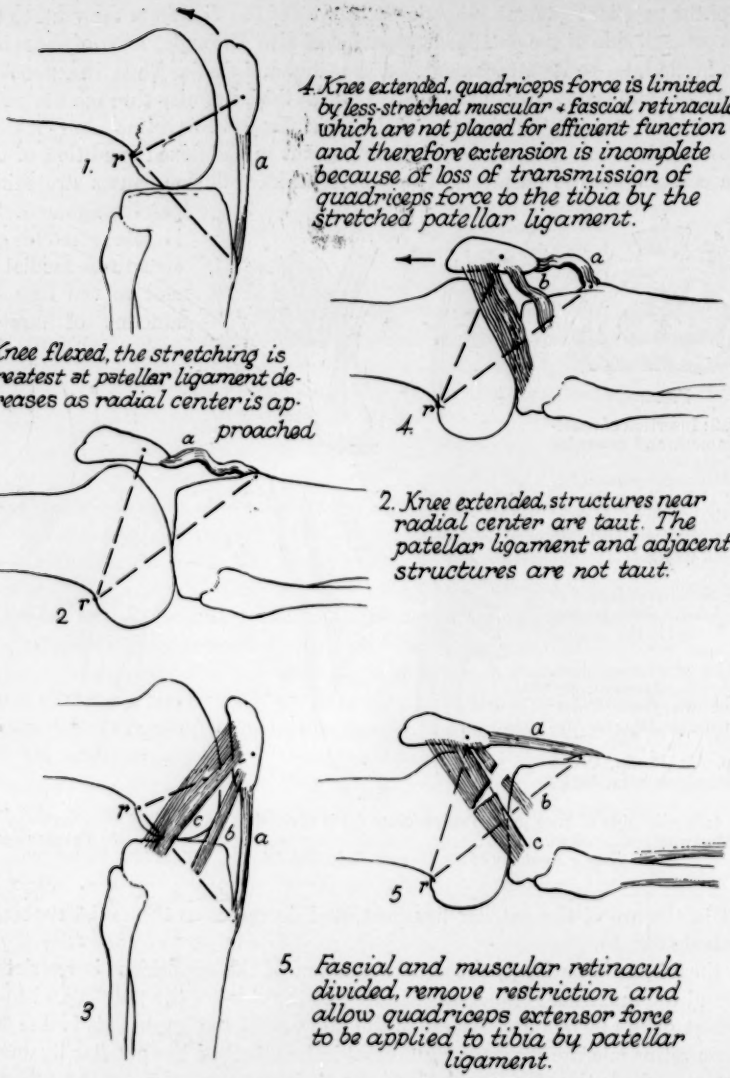
1. Knee flexed, the stretching is greatest at patellar ligament decreases as radial center is approached
r
a
4. Knee extended, quadriceps force is limited by less-stretched muscular + fascial retinacula which are not placed for efficient function and therefore extension is incomplete because of loss of transmission of quadriceps force to the tibia by the stretched patellar ligament.
a
b
r
4.
a
2. Knee extended, structures near radial center are taut. The patellar ligament and adjacent structures are not taut.
r
2
r
c
b
a
3
a
b
c
r
5
5. Fascial and muscular retinacula divided, remove restriction and allow quadriceps extensor force to be applied to tibia by patellar ligament.
3. Structures near radial center which are less-stretched are fascial retinaculum and lateral and medial portions of muscular retinacula
a.-patellar ligament
b.-muscular retinaculum
c.-fascial
r.-radial center

FIG. 5

Diagrammatic representation of patellar extension, restrictive structures.

line as the fascial retinaculum (Fig. 6). After this has been performed, the capsule of the joint is visible, but it is not incised; therefore, the knee joint is not opened.

The procedure is then carried out on the medial side of the knee through a three-inch incision, one-half inch medial and parallel to the patella and the patellar ligament; the incision is started at the same distal level as the lateral incision and proceeds proximally. The fascial retinaculum is divided in the line of the incision and then is curved posteriorly to a point at the level of the femoral attachment of the tibial collateral ligament. The next

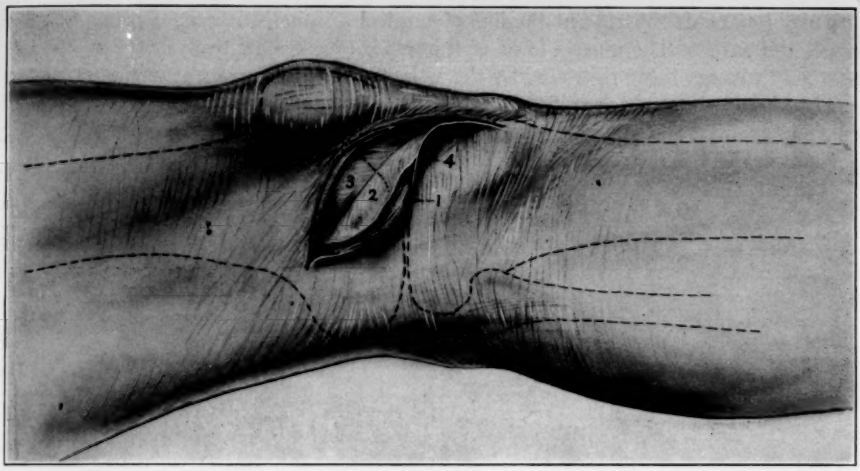

FIG. 6

The skin and tela subcutanea have been removed. Retinacular structures are divided surgically on the lateral aspect of the knee joint. Incisions must go down to the level of the anterior margin of the fibular collateral ligament.

1: Fascial retinaculum. *2:* Muscular retinaculum.
3: Capsule of joint. *4:* Fusion margin of musculofascial retinacula.

structure seen is the muscular retinaculum, and this is divided in the same line as the fascial retinaculum. As before, the capsule of the joint is visible, but it is not opened (Fig. 7).

The skin incisions are closed by silk sutures. One must remember that the fascial and muscular retinacula are fused, distal to the patella. The incisions in the retinacula are not sutured. If the surgeon wishes, he may curve the skin incisions posteriorly at the proximal ends.

When the anaesthetic has been discontinued, the muscle tone of the quadriceps physiologically causes tension on the patellar ligament, because the divided fascial and

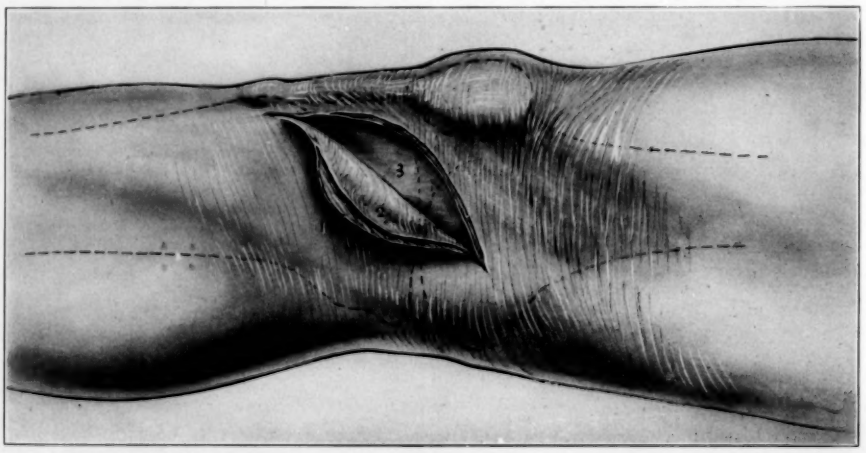

FIG. 7

The skin and tela subcutanea have been removed. Retinacular structures are divided surgically on the medial side of the knee joint. Incisions must go down to the level of the anterior margin of the tibial collateral ligament.

1: Fascial retinaculum. *2:* Muscular retinaculum. *3:* Capsule of joint.

muscular retinacula separate at the line of surgical division, with slight proximal migration of the patella. The muscle force of the quadriceps is now transmitted to the tibial tuberosity without being restricted by the retinacula, and complete extension of the knee joint by the quadriceps is possible. The slight relative change in the position of the patella is not important. As healing occurs, the retinacula unite in their new relationship and the previous stability is restored (Figs. 8-A, 8-B, and 8-C).

RESULTS

The advantages of this procedure are its simplicity, the short duration of healing time, early ambulation, and physiological shortening of the quadriceps. The method avoids

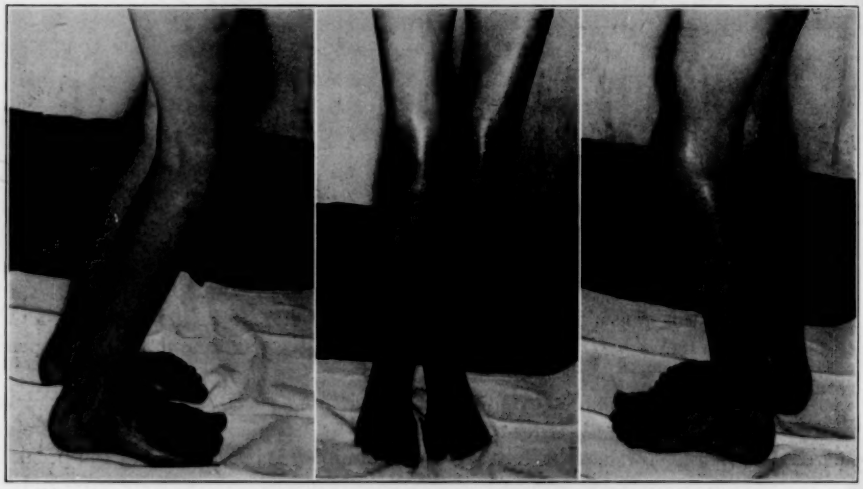

FIG. 8-A

FIG. 8-B

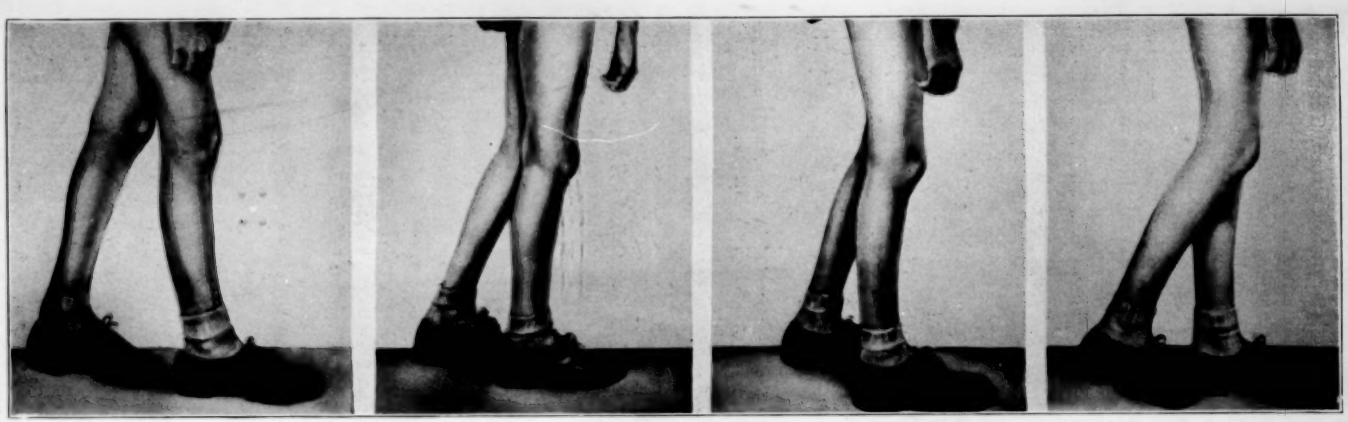

FIG. 8-C

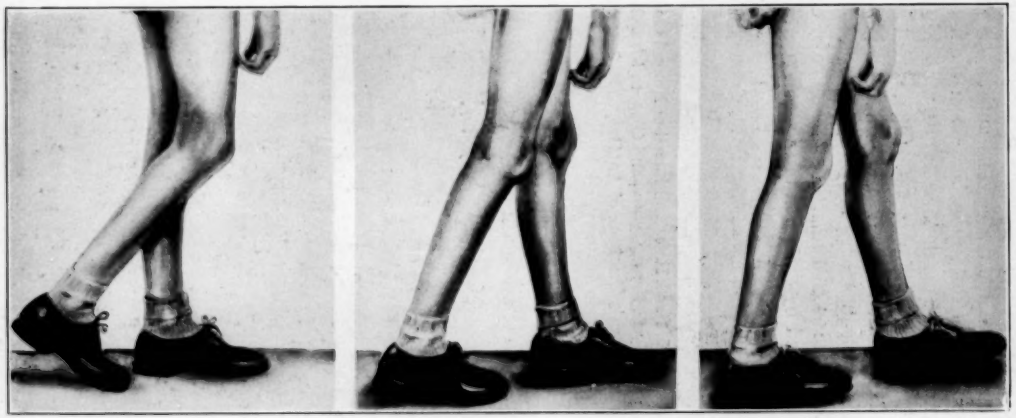

FIG. 8-D

Fig. 8-A: Right lateral, anterior, and left lateral views of knees before operation. Note musculofascial tension in the suprapatellar area.

Fig. 8-B: Right lateral, anterior, and left lateral views of knees after operation. Anterior scars indicate retinacula release; posterior scars, hamstring releases.

Figs. 8-C and 8-D: Retouched enlargements of motion pictures, showing the walking gait eight months after operation.

failure of fixation from the pulling out of screws or the breaking of sutures or wires; in addition, damage to the tibial epiphysis is avoided, since the tibial tubercle is not disturbed.

Evaluation of the patient's ability to become ambulatory is necessary. One must remember the limitations of the spastic individual in anticipating the result desired.

During the period from 1937 through 1948, the retinacula release was performed in sixty-three selected cases, consisting of seven hemiplegics and fifty-six diplegics. Of the seven hemiplegics, four had satisfactory results and three did not. The diplegic group had forty-seven satisfactory results and seven unsatisfactory results; one complete failure occurred because the hamstring muscles were not released, and there was also one case of recurvature of the knees. In the case of recurvature, neurectomy of the branches of the gastrocnemius heads had previously been performed. We do not use this operation, preferring the soleus neurectomy for tendo achillis tension, and this was an error in selection of the case.

The hamstring release has varied from lengthening to transfer of the tendons to the condyles of the femur. The author has preferred transplantation of the tendons to the condyles of the femur, because of the favorable effect on extension of the hip and of the knee joint. This method has been used in all of the recent cases.

The time of observation has varied from three months to ten years. The best period of evaluation after operation is probably about one year after release of the retinacula. The patients ranged in age from seven to seventeen years; a little more difficulty was experienced in teaching those in the older age group maintenance of the corrected posture permitted by extension of the knee joints.

NOTE: The author wishes to express his appreciation to the Department of Anatomy, the Department of Medical Illustration, and Miss Estelle Greenwalt, University of Texas School of Medicine.

REFERENCES

CHANDLER, F. A.: Re-establishment of Normal Leverage of the Patella in Knee Flexion Deformity in Spastic Paralysis. Surg., Gynec., and Obstet., **57**: 523-527, 1933.

CUNNINGHAM'S Textbook of Anatomy, Ed. 8, pp. 367 and 513. New York, Oxford University Press, 1943.

MORRIS' Human Anatomy, Ed. 10, p. 537. (Edited by J. P. Schaeffer.) Philadelphia, The Blakiston Co., 1942.

POTT'S PARAPLEGIA*

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Prolonged rest, as the treatment of paraplegia complicating tuberculosis of the spine, has been axiomatic in the past. A review of the authors' patients with Pott's paraplegia appeared worthy of report. The pathological findings at operation and at three post-mortem examinations suggested that prolonged rest often had not been sufficient to effect the desirable end result. The late end results also were disappointing. This study is based upon an analysis of fifteen children and seventeen adults, suffering from this distressing disability.

Sorrel, Girdlestone, Seddon, and Butler have written excellent articles on the subject. Sorrel was the first to differentiate between early-onset and late-onset paraplegia, and complete and incomplete paralysis.

For the purpose of discussion, the following descriptive terms are used:

1. *Paraplegia in extension* indicates the minimal involvement of the corticospinal pathway, causing muscle spasticity with the lower limbs in extension.

2. *Paraplegia in flexion* indicates more complete corticospinal involvement, resulting in severe muscle spasm with the lower limbs in flexion.

3. *Paraplegia with flaccidity* indicates complete involvement of both motor and sensory pathways of the spinal cord. The muscles are flaccid because of loss of reflex activity. As the cord shock subsides, painful flexor spasms develop.

4. *The spine-tumor syndrome* is so called because the neurological signs of paraplegia appear before there is roentgenographic evidence of tuberculosis of the vertebrae. This is a rare condition; in this series, it was seen but once, with early tuberculosis of the spinous process.

Causes of Paraplegia

The symptoms of Pott's paraplegia become apparent when there is interference with the conductivity of the spinal cord. This may be caused by the inflammatory process, resulting in oedema of the cord, pressure of granulation tissue, tension abscess, or tuberculous pachymeningitis. Microscopic evidence of pachymeningitis was found in two patients. Mechanical pressure from sequestra and pathological dislocation of the spine was also observed. In pathological dislocation of the spine, the stretching of the cord over a sequestrum of bone, over an intervertebral disc, or over the posterior portion of the body of the vertebra, can be demonstrated. The pressure is greater directly over the anterolateral columns, so that motion is affected earlier than is sensation.

Early-Onset Paraplegia

In early-onset paraplegia, the signs appear early, usually during the first two years of the disease. The paralysis may appear even before the diagnosis of tuberculosis of the spine has been made. The palsy is usually more complete than in the late-onset type. The authors believe the symptoms are caused by inflammation, oedema, or abscess formation. Therefore, rapid and complete recovery usually follows efficient recumbency treatment. Paraplegia of this type is common and requires no surgery for relief, unless, while under conservative treatment, it progresses, showing symptoms of the late-onset type.

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 25, 1949.

Late-Onset Paraplegia

Late-onset paraplegia occurs from two to many years after the onset of the spine lesion. The disease process either has never been quiescent or reactivation has occurred. The paralysis is often incomplete, selective, and progressive. Girdle pains may be the first complaint, although loss of coordination in the lower extremities may be the initial symptom. The tendon reflexes are hyperactive. Loss of sensation appears late in the progress of the disease and may be patchy. Loss of sphincter control is a later development in the disease. The paralysis is evident with the extremities in extension. Rarely does this type progress to the so-called paraplegia in flexion. The slightest stimulation will cause the lower extremities to flex vigorously at the knees and hips, which may be attended with much pain. It may become difficult to hold down the lower extremities. Anaesthesia of the lower extremities is more diffuse, and loss of vibratory sense is apparent. The condition of the patient becomes rapidly worse, and there is danger of permanent paraplegia. The prognosis is then grave. Certainly surgical intervention should be contemplated before the paraplegia has progressed to this stage, especially if partial or complete spinal-fluid block has been demonstrated.

Study of the Spinal Fluid

The spinal fluid and its dynamics were studied in eight patients. In three of these, a manometric block was revealed; one patient had a partial block which was further substantiated by normal spinal-fluid dynamics at the cisternal level. The highest spinal-fluid protein was 1251 milligrams per 100 cubic centimeters; the lowest was 233 milligrams per 100 cubic centimeters.

Sites of Spinal Lesions

The sites of the lesions of the spine were all in the thoracic or thoracolumbar vertebrae, except in one child with tuberculosis of the cervical vertebrae.

Of twenty-two patients treated by recumbency, eleven completely recovered, three partially recovered, three remained paraplegic, and five died. The longest time required for complete recovery was twelve months; the shortest period was two months. The signs of recovery usually appear within three or four months; if they do not appear within this time, a renewed effort should be made to determine if the indications for surgical intervention are present.

PATHOLOGICAL FINDINGS

Pathological Findings at Operation

Laminectomy was performed upon seven patients. A tuberculous granuloma was found in four cases, and complete recovery followed.

One patient, whose condition had been unimproved, had a large abscess drained. This patient also had a very thick dura. The surgery was done four years after the onset of the paralysis. In another, a large abscess and a sequestra were found. This patient died and, at autopsy, an area of compression of the cord was found at the site of a pathological dislocation of the spine.

Pachymeningitis was demonstrated in one case at laminectomy. In no case did the authors fail to demonstrate a cause for the paralysis.

Costotransversectomy was performed upon four patients, with complete recovery in three instances. The fourth patient died seven months after the operation, in a state of partial recovery, although no cause for the paralysis had been found. Abscesses were found in three cases.

Pathological Findings at Postmortem Examination

Careful postmortem examinations and studies were made of three patients who died

in a paraplegic state. Eleven patients who had been subjected to surgical exploration were also observed. The pathological findings and observations indicated that paraplegia may be attributed to two factors, mechanical and inflammatory.

The mechanical factors are (1) encroachment on the cord by tuberculous tissue; (2) direct local pressure by a paravertebral abscess; (3) pressure from a sequestrum; and (4) pathological fracture-dislocation at the kyphos.

The inflammatory causes may be (1) toxic; (2) vascular reaction, secondary to the disease; and (3) pachymeningitis.

Figure 1-A shows, in a gross specimen, an area of local pressure on the spinal cord, caused by an extruded sequestrum. Vertebral-body collapse with anterior angulation of 15 to 20 degrees is noted. There is also evidence of growth of tuberculous granulation tissue into the vertebral canal and its impingement upon the dura and the spinal cord. Microscopic section of the dura at this level reveals invasion by the tuberculous granulation tissue, which indicates the existence of local pachymeningitis. The authors agree with Seddon and Butler that the dura acts as a barrier, by hyperplasia, but the authors believe that pachymeningitis may and does occur. Pachymeningitis was seen in two patients, and was proved by microscopic sections in both instances. The cord section of Figure 1-A reveals vacuolization and fenestration, with gitter-cell formation in the thoracolateral columns of the cord,—a pathological process indicative of cord softening. The pathological cause of the paraplegia may be pressure from a sequestrum, and tuberculous granulation tissue infiltrating the dural covering of the spinal cord.

FIG. 1-A

Case 1. Gross specimen shows pressure by a sequestrum and growth of granulation tissue into the vertebral canal.

Another gross specimen (Fig. 2-A) reveals typical vertebral destruction. The posterior portion of the spine is the site of a previous laminectomy, at the lower margin of which is a definite encroachment on the spinal canal, causing marked diminution in the caliber. The microscopic sections of the spinal cord (Fig. 2-C) at the level of the constriction reveal a compression myelitis. No distinction is evident between the gray and white matter. High-power fields reveal numerous astrocytes and gitter cells. The proliferation of astrocytes is due to marked myelin destruction, and the gitter-cell accumulation is due to phagocytosis of cellular debris. There is no abnormality of the cord or dura at the apex of the kyphos. A sequestrum, which was believed to be causing the pressure, had been removed at laminectomy. Had the laminectomy been continued caudally, more complete relief of pressure would have been obtained. The cause of the paraplegia in this patient is believed to have been purely mechanical.

Figure 3-A, a gross specimen, is an example of typical vertebral-body involvement. The destruction is not so advanced as in the previous specimen. The spinal canal is smooth throughout. The only pathological finding in the sections of the cord and its coverings was an adherent piarachnoid. The cause of the paraplegia may be assumed to be

FIG. 1-B

A section of the dura, showing tuberculous pachymeningitis (× 8.3).

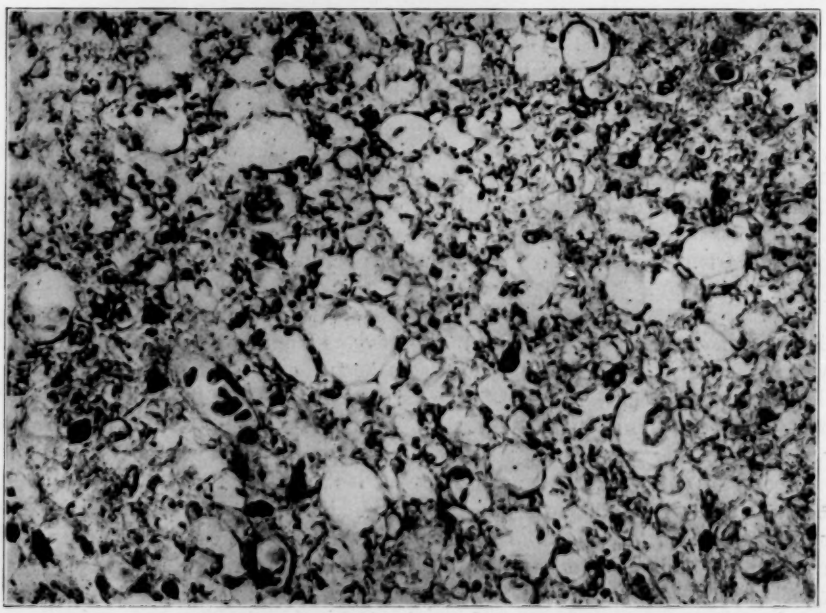

FIG. 1-C

A section of the cord, with softening, gitter-cell formation, vacuolization, and fenestration (× 500).

inflammatory in type, probably an oedema of the cord covering. As the inflammatory reaction subsided, the adhesions were the only residuum. The patient died of tuberculous

FIG. 2-B

Walling off of the dura by the inflammatory reaction, thus protecting the meninges (× 8.3).

FIG. 2-A

Case 2. Gross specimen shows encroachment on the spinal canal below the level of the laminectomy. Sequestrectomy had been performed one year previously.

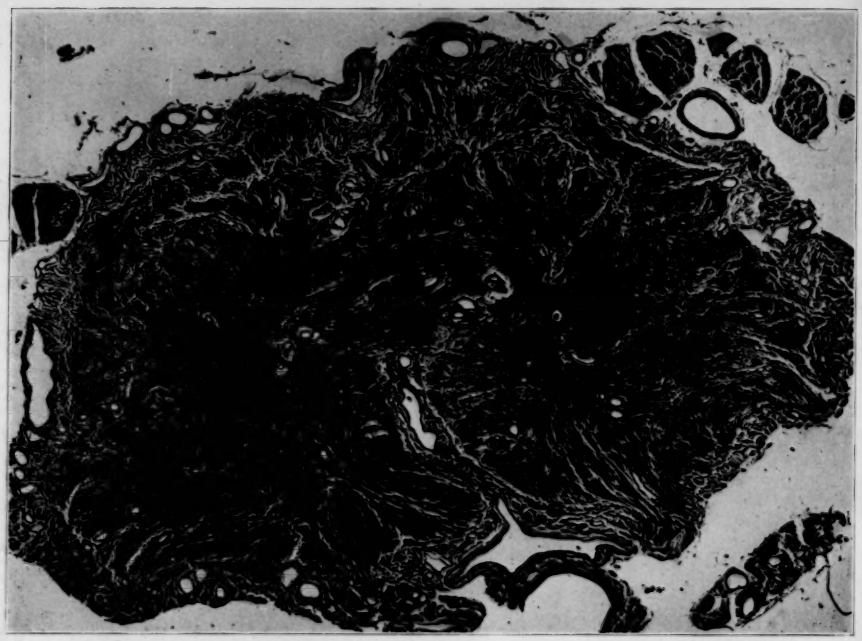

FIG. 2-C

Compression myelitis resulting in loss of distinction between gray and white matter in the spinal cord (× 8.3).

pericarditis six weeks after spine fusion, but eighteen months after complete recovery from his paraplegia.

INDICATIONS FOR SURGERY

When conservative treatment of three or four months' duration fails to give signs of beginning recovery, surgical indications should be sought and the route to follow should be determined. Spinal-fluid block may be the deciding factor.

Laminectomy is indicated in tuberculosis of the vertebral appendages and when there is minimal evidence of a tension abscess or of bone pressure. It may also be indicated following a non-productive costotransversectomy. A tuberculous vertebral appendage, a granuloma, or a caseous abscess within the spinal canal is easily exposed and removed. The authors have recently observed two patients with tuberculosis of the vertebral appendages, who have made rapid recovery following laminectomy.

Costotransversectomy is indicated when there is evidence of a tension abscess or a sequestrum. The abscess is drained away from, rather than around, the spinal cord.

If the spine has not been fused previously, fusion may be done at operation or afterward. Eleven of the thirty-two patients had had spine-fusion operations previous to the onset of the paraplegia.

SUMMARY

From a prolonged and careful study of the small series here reported, it is evident that Pott's paraplegia can be simply classified as early-onset and late-onset types. The changes which occur in the two types need not necessarily change the classification of a given case, but a case of early-onset type may become like one of late-onset type.

The prognosis is always considered grave, until signs of recovery appear during recumbency treatment.

Patients with Pott's paraplegia should not be allowed to remain in a paraplegic state indefinitely. Studies of the dynamics of the spinal fluid and other studies should be made to determine whether surgery is indicated.

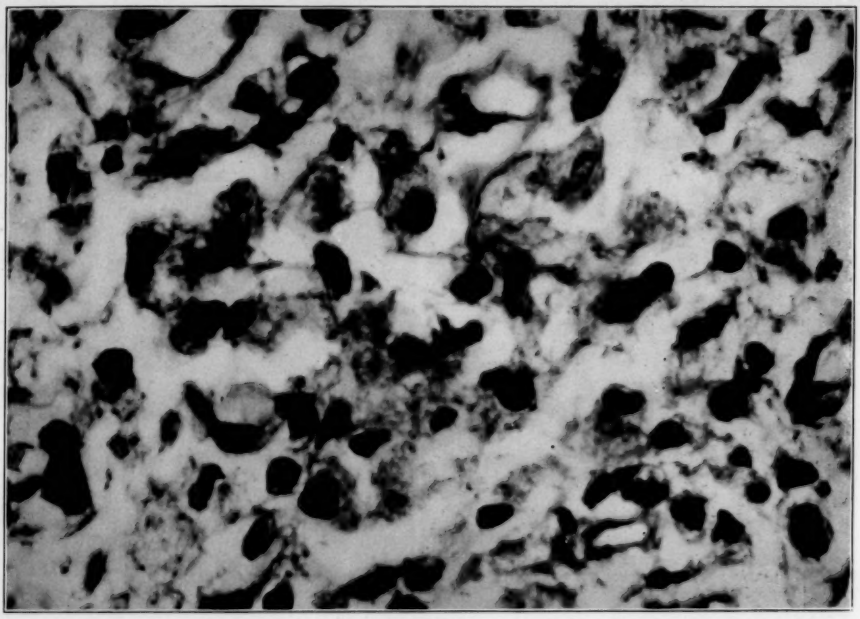

FIG. 2-D
Gitter-cell formation (× 667).

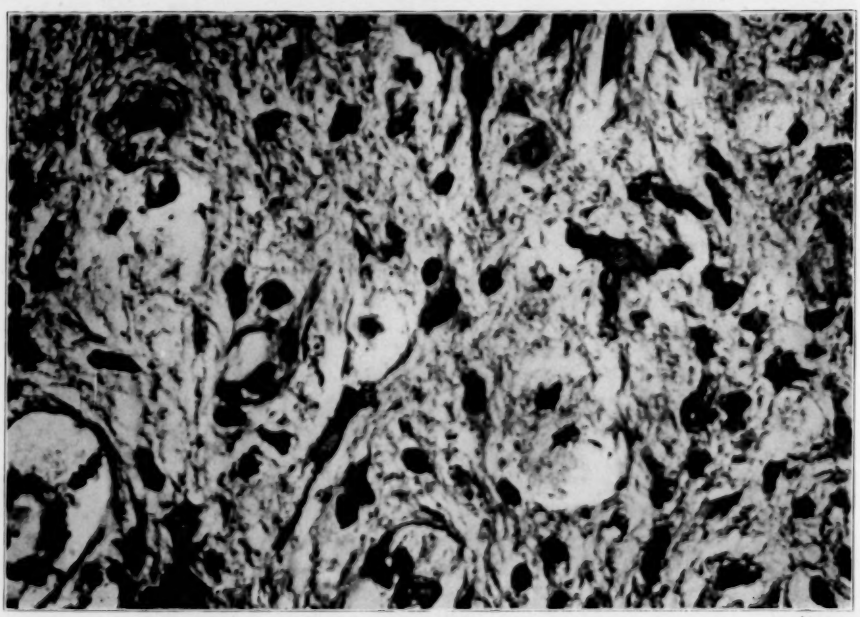

FIG. 2-E
Astrocyte proliferation due to myelin destruction (× 667).

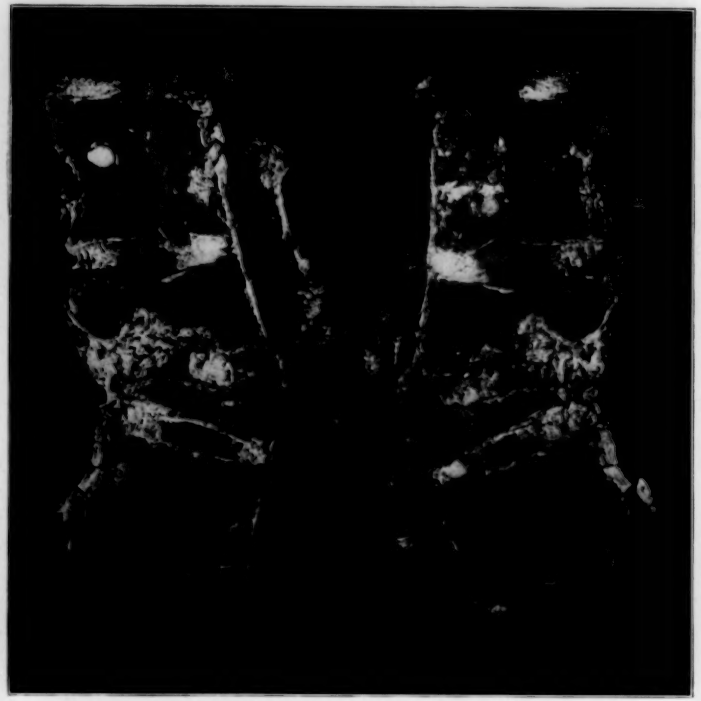

FIG. 3-A

Case 3. Typical vertebral-body involvement. The spinal canal is smooth. There has been no compression.

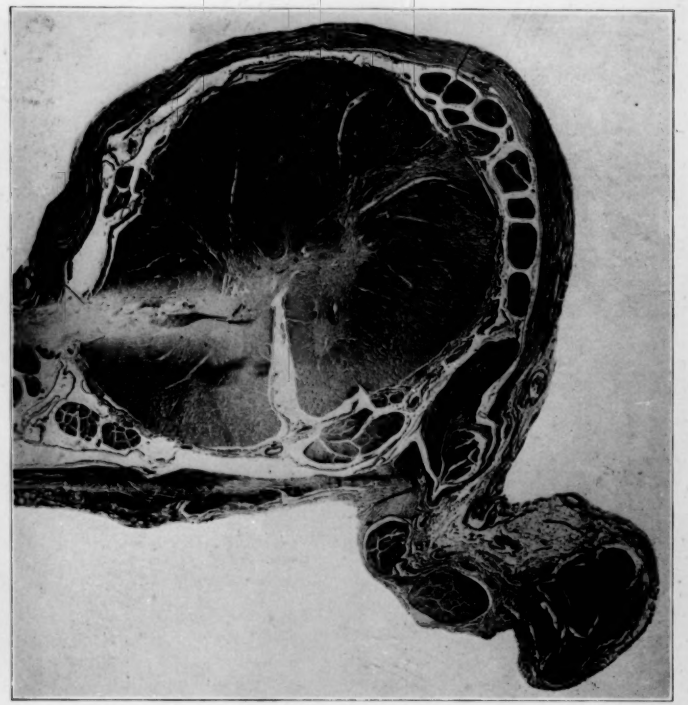

FIG. 3-B

A normal cord is shown, but there is an adherent piarachnoid, believed to be secondary to the inflammatory process (× 8.3).

Of six patients operated upon less than nine months after the onset of the paraplegia, five completely recovered and one died.

Of four patients operated upon one or more years after the onset of the paraplegia, two died, one completely recovered, and one is unimproved. Patients operated upon fairly early, in whom conservative treatment had failed, recovered more rapidly and completely than those operated upon later. Of twenty-two patients treated by recumbency, eleven completely recovered; three were unimproved; three recovered partially; and five died.

No ill-effects or deaths can be attributed to the two surgical procedures employed.

NOTE: The authors wish to express their sincere thanks to A. C. Michael, M.D., of the Department of Pathology, Indiana University School of Medicine.

REFERENCES

BUTLER, R. W.: Paraplegia in Pott's Disease, with Special Reference to the Pathology and Etiology. British J. Surg., **22**: 738–768, 1935.

GIRDLESTONE, G. R.: Tuberculosis of Bone and Joint. London, Oxford University Press, 1940.

SEDDON, H. J.: Pott's Paraplegia: Prognosis and Treatment. British J. Surg., **22**: 769–799, 1935.

SORREL, ETIÉNNE, et SORREL-DEJERINE, MME.: Les formes anatomo-cliniques des paraplégies Pottiques. Leur évolution—leur pronostic. Presse Méd., **34**: 785–788, 1926.

DISCUSSION

DR. MATHER CLEVELAND, NEW YORK, N. Y.: Percival Pott presented, in 1779, his "Remarks on That Kind of Palsy of the Lower Limbs, Which Is Frequently Found to Accompany a Curvature of the Spine, and Is Supposed to Be Caused by It. Together With Its Method of Cure" (*In* Miscellanea Medica, *V*, London, 1779). He stated that this paralysis attacked both sexes and all ages, adding: "This in an infant is melancholy to see, in an adult most miserable to endure". He denied that the curvature of the spine exerted any influence on the palsy, stating categorically that an abscess was responsible and that its drainage usually brought relief from the symptoms. He cited a passage from Hippocrates in which this ancient physician described paralyses of the lower limbs cured by an abscess in the back or loins.

Pott did not wait for spontaneous drainage of the abscess to occur. He performed surgical drainage by means of cauterization and setons (retained foreign bodies of silk thread). The cause of this distressing complication of tuberculosis of the spine, and measures to relieve it,

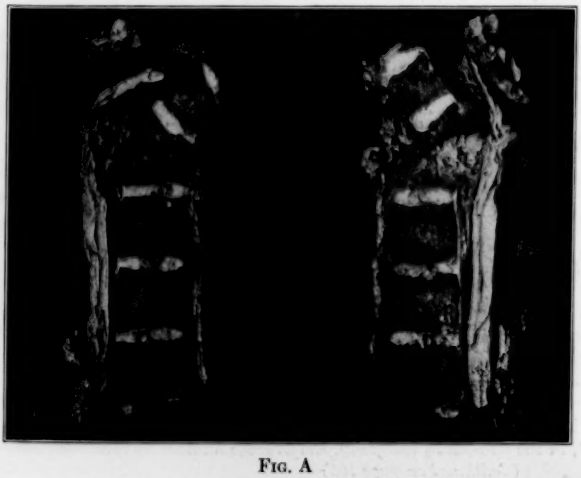

FIG. A

FIG. B

Fig. B has been reproduced, by permission, from *American Review of Tuberculosis.*

were clearly presented almost two centuries ago. In ending his essay, Pott stated: "what I have affirmed, is what I have seen and proved, and that the objections are merely speculative and theoretical".

Our experience with paraplegia complicating tuberculosis of the spine at Sea View and St. Luke's Hospitals extends over a period of twenty-two years. A study of 203 cases of tuberculosis of the spine treated surgically at those Hospitals was reported in 1940. Symptoms of compression of the spinal cord were noted in forty-eight patients, an incidence of 23.5 per cent. Seventeen of these had slight transitory spasticity of the lower extremities, from which they recovered uneventfully with continued bed rest. Of thirty-one patients who had complete paraplegia, the complication developed in thirteen prior to spine fusion, and in eighteen the symptoms appeared after operation. The mortality rate among these paraplegics, due to tuberculosis of the spine, was 42 per cent. Of the eighteen survivors, all but one recovered motor power, and the disease was arrested. We still feel that a successful surgical fusion of the spine and prolonged bed rest offer the best means of relieving this serious complication.

Laminectomy was performed on a number of patients prior to 1930, with dramatic relief in one or two instances. This procedure has seemed rather futile to us, for the most part, because it fails to relieve the pressure on the ventral and lateral surfaces of the spinal cord which is usually exerted by the abscess. Laminectomy renders difficult the surgical stabilization of the diseased spine which is so essential to the ultimate arrest of the process.

Two pathological specimens of paraplegia in tuberculosis of the spine have been selected:

Figure A shows a spine with collapse of two mid-thoracic vertebrae; an abscess filled with debris is impinging upon the spinal cord. This patient had a laminectomy following fusion of the spine without relief of his symptoms; he died within a few months. This occurred a good many years ago. Anterior decompression of the abscess, which was not practised at that time, might have helped materially.

Figure B shows a patient who had complete paraplegia; efforts to locate the site of the disease by roentgenograms were unsuccessful. The entire spine is invaded without collapse of a vertebra and with no thin joint space. The abscess surrounded and impinged upon a large segment of the spinal cord. Decompression could only have been accomplished by drainage of this mediastinal abscess.

I am in full agreement with Dr. Garceau and Dr. Brady that these paraplegics should not be allowed to remain paralyzed indefinitely, if means can be devised to help them. Drainage of a psoas, lumbar, or mediastinal abscess by aspiration or incision should be performed to relieve spinal-cord pressure. By the use of streptomycin as an adjuvant, probably a good many of these abscesses may be incised and drained, with perhaps immediate closure of the wound.

Anterior decompression in disease of the thoracic spine, to relieve the pressure of an abscess and bone detritus, or both, should be preceded by spine fusion. The experience at Sea View Hospital with this procedure has been limited, and it has, for the most part, been performed in cases of long duration.

Of five patients subjected to anterior decompression, four recovered approximately 50 per cent. of their motor power and one died of tuberculosis of the meninges. If this type of operation is to help, it should be employed much earlier, as Dr. Garceau and Dr. Brady have suggested.

I would like to congratulate the authors for re-emphasizing and reaffirming certain known facts regarding this alarming complication of tuberculosis of the spine.

DR. J. HIRAM KITE, ATLANTA, GEORGIA: I wish to thank Dr. Garceau and Dr. Brady for bringing to us an old and familiar subject in a program containing so many new topics. I would like to question the advisability of using the title "Pott's Paraplegia". As Dr. Cleveland has said, it was in 1779 that Pott first described this condition. Pott didn't know what caused tuberculosis of the spine. It was 100 years before the tubercle bacillus was isolated, but during that century the doctors referred to the syndrome as "Pott's Paraplegia". Now that the diagnosis has been accurately established, it seems wiser to use a descriptive term, instead of a man's name, and to call it "tuberculosis of the spine with paraplegia".

As Dr. Cleveland has said, Pott kept sores on these patients' backs by running a seton under the skin, by repeated incisions, or by caustics. He thought he was draining off the inflammation; however, since the sore was on the back, the patient had to stay in bed on the abdomen, and it was the bed rest which helped the inflammation. What is good for patients with tuberculosis of the spine is also good for patients with paraplegia. Dr. Ridlon, in 1916, called attention to the fact that Pott was not the first to describe this condition. Jean Pierre David had given a better description of it a year before.

In a comparison * of eighty-nine children with tuberculosis of the spine without paraplegia, and twenty cases of spinal tuberculosis with paraplegia, the patients in whom paraplegia might develop may be selected from the group. We have found that the area most often involved in the non-paralytic group was from the ninth thoracic to the second lumbar vertebra. In the paralytic group, the areas most frequently involved were from the fifth to the ninth thoracic vertebra. Anatomists tell us that the spinal canal is smallest at this level; therefore, the products of inflammation would cause the maximum pressure on the cord at this level.

From this study, we note that, in the non-paralyzed group, the average number of vertebrae involved

(*Continued on page 102*)

* Table 2 *in* Tuberculosis of the Spine with Paraplegia. Southern Med. J., **29**: 883–891, 1936.

INTERVERTEBRAL-DISC LESIONS IN CHILDREN AND ADOLESCENTS

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In spite of the fact that low-back pain, with or without sciatica, is a frequent complaint and cause of disability in adults, this condition is rarely encountered in children or adolescents. When pain in the low back is encountered in a child or adolescent, one expects to find roentgenographic evidence of disease, especially tuberculosis, neoplasm, or epiphysitis.

In adults, on the other hand, the low-back pain and sciatica are, in the great majority of instances, idiopathic. By idiopathic we mean that the symptoms arise in the low back and are not caused by a specific disease, such as tuberculosis or a neoplasm. For reasons which have been given elsewhere [2,3], it is believed that, in nearly all of these patients with idiopathic low-back pain, the symptoms are caused by lesions of the intervertebral discs in the lumbosacral region (third, fourth, and fifth lumbar discs). The author believes that, in most cases of idiopathic low-back pain in children and adolescents, the symptoms are caused by intervertebral-disc lesions.

However, the fact that intervertebral-disc protrusions may occur in children and cause symptoms is not generally recognized. Of the hundreds of papers which have been written on intervertebral discs, only one has been found in which a protruding intervertebral disc in a child was described [5]. This is largely because what is now recognized as the typical lumbosacral-disc syndrome is rare in children and adolescents. That it may occur is evidenced by the report by Wahren of a case in a girl, twelve years of age.

Pain developed in this patient's back after she had been standing on her hands, with her back arched and her feet against the wall. The pain gradually became worse and extended down the right sciatic distribution to the heel. It was increased by coughing, sneezing, or activity, and was relieved by rest in bed. When examined, the patient was unable to walk upright. There was spasm of the lumbar muscles and a list to the left. The Lasègue sign was positive with the lower extremities elevated 10 degrees bilaterally. The ankle jerk was absent on the right. Sensation was normal. Roentgenograms and lumbar puncture revealed nothing abnormal.

There was no improvement after rest in bed for one month. At operation, a pea-sized protrusion of the fifth lumbar disc was found beneath the first sacral nerve. When incised, the contents of this nodule were found to be under pressure and somewhat more mucous than is usual in disc protrusions. The patient was relieved of pain by the operation.

That such patients have been seen by others is evidenced by the report of Love on 1,217 patients operated upon for ruptured intervertebral discs at the Mayo Clinic in a three-year period. In his series, twenty-five or 2.1 per cent. of the patients were between the ages of ten and nineteen years.

The author has operated upon four patients of this type,—a girl twelve years of age, a girl fourteen years of age, and two boys eighteen years of age. All were severely disabled with low-back pain and sciatica and presented the typical disc syndrome, which included limitation of movement in the low back, pain on deep pressure over the pathological disc, and tenderness of the sciatic nerve or of some of its branches, in addition to the findings mentioned by Wahren. None of these patients had obtained relief by conservative treatment which, in three cases, had included rest in bed for a month or more. The fourth patient was operated upon about two weeks after the onset of symptoms, because it had not been possible to relieve the severe pain by conservative treatment, including rest in bed and manipulation.

All of these patients were operated upon without preliminary myelography. In each, a large, bulging fifth-lumbar disc was found beneath the first-sacral root. When this dome-like swelling was incised, some grayish-white gelatinous material exuded into the spinal canal. Similar material was removed from the interior of the disc. This material resembled the substance of the normal nucleus pulposus. There were no sequestra, and the only pathological change noted was the thinning and bulging of the posterior wall of the annulus fibrosus on the affected side.

Since one of these patients was first seen at the age of fourteen years and was operated upon four years later, the case will be reported in detail.

Case Report

A muscular boy, fourteen years of age, complained of pain in the low back and right lower extremity. The pain began after he had taken part in a weight-lifting contest, and was localized in the low back. He had had several osteopathic treatments with temporary relief, in the beginning of his illness; but the alleviation rarely lasted more than a few days, and later treatments had failed to relieve the pain even temporarily. The pain became more severe and gradually extended down the back of the right thigh and leg.

When the boy was first seen by the author, the pain in the low back was quite severe and was aggravated by coughing, sneezing, straining at the stool, prolonged standing, or physical activity. The patient stated that he was unable to walk more than 100 yards without stopping to rest, and that he usually squatted down to lessen the pain. The pain was relieved by lying down and did not keep him awake at night. There had been no numbness, tingling, or weakness of the leg. At the time of the first examination, he was not having any leg pain.

On physical examination, the patient was a strong, healthy-appearing boy. He walked with a peculiar gait in that the spine was relatively stiff, and he swayed backward from the hips as either the right or left foot was thrust forward and the knee was straightened. As he stood, his back was relatively flat with moderate muscle spasm; forward flexion of the spine was markedly limited and was accompanied by pain in the low back. Other movements of the back were fairly free and were not accompanied by pain. With the patient recumbent, straight-leg raising was limited to about 20 degrees bilaterally and was accompanied by pain in the lumbosacral region. With the knee flexed, movements of the hips were free and painless. On deep palpation, there was moderate localized tenderness over the lumbosacral disc (between the spines of the fifth lumbar and first sacral vertebra). Knee jerks and ankle jerks were normal, and no sensory disturbance was noted. Roentgenograms of the lumbosacral spine disclosed nothing abnormal.

The patient was forbidden to engage in athletics and was put on a regimen of relative rest; he was advised to sleep with a fracture board underneath the mattress, and was given a wide lumbosacral belt for support and in order to limit movement in the low back. As his symptoms subsided, he was given exercises to strengthen his abdominal and back muscles; and after several months he was able to resume athletics.

He got along well for four years. Then, while engaged in a wrestling bout with another student, severe pain developed in the low back. This pain, which quickly extended down the back of the right leg, was excruciating in character and was not relieved by rest in bed. After the patient had spent several days in bed and had endured sleepless nights, he was brought to the Hospital. As we were not successful in relieving his pain by conservative measures, he was operated upon and the vertebral canal in the lumbosacral region was explored. A large protrusion of the fifth lumbar disc was found on the right side, beneath the root of the first sacral nerve. When the swelling was incised, some grayish-white gelatinous material exuded; more was removed from the disc cavity.

The pain was relieved promptly; the patient left the Hospital on the eighth postoperative day and resumed his class work about a week later. Recovery was complete and he resumed athletics gradually; to date (six years later) there has been no recurrence of the pain.

This case is described in detail because it convinced the author that many of the children and adolescents from eight to eighteen years of age (of whom we see six or more a year on account of low-back pain, sciatic pain, or both, and disability) are suffering from lesions of one or possibly more intervertebral discs in the lumbosacral region. During the period of observation, this patient's symptoms varied greatly while the disc lesion was causing trouble, and his physical findings varied from those of a boy who presented nothing abnormal except slight localized tenderness over the lumbosacral disc to one who presented a typical severe acute disc syndrome.

CLINICAL FINDINGS

While children and adolescents may present a fairly typical disc syndrome with

symptoms which are sufficiently severe and intractable to warrant operative treatment, this is the exception and not the rule. (In a series of over forty such cases, only four patients have been operated upon.) In the majority of instances, the symptoms are intermittent and relatively mild, and the parent is more alarmed than the patient. The pain may have subsided by the time the patient reaches the doctor's office, and it may be difficult to obtain a history of pain and disability in the low back. This is especially true if the patient is anxious to engage in competitive athletics.

The patient may be referred because of pain, a limp, ease of fatigue, difficulty in sleeping, or trouble in stooping. If he is on an athletic team, it is not unusual to find that he has been treated with local heat and massage or has been referred to an osteopath or chiropractor by his coach or trainer. The physical signs may be more marked than are the complaints (just the opposite of what one usually finds in adults with similar lesions).

These disc lesions seem to occur more frequently in boys than in girls, and may or may not follow a specific injury. In some cases, the symptoms begin gradually in boys or girls who are engaged in competitive athletics. The pain may be unilateral or in the mid-line and, in severe cases, may extend into the buttock, thigh, or leg. Rarely is it aggravated by coughing, sneezing, or straining at stool. Usually it is worse in the evening; it is aggravated by activity (especially stooping, lifting, or prolonged standing) and relieved by rest in bed. None of the author's patients have complained of numbness, paraesthesia, or muscle weakness.

The three positive physical findings most frequently encountered are:

1. Pain on deep pressure over the fifth lumbar disc,—between the spines of the fifth lumbar and first sacral vertebrae. (The author has not diagnosed a lesion of either the fourth or third lumbar disc in children or adolescents.) In a mild case, this localized tenderness may be the only abnormal finding at physical examination; but, with a satisfactory history and a negative roentgenogram, it is believed that this is sufficient to warrant a tentative diagnosis of an intervertebral-disc lesion in this area. In a patient who is recovering as a result of conservative treatment, this lumbosacral tenderness is the last sign to disappear.

2. Limitation of straight-leg raising on one or both sides. This may be present in patients who have not had sciatic pain, and the limitation may or may not be accompanied by pain in the low back or lower extremity at the limits of movement. The limitation may be slight or marked, and tends to disappear as the patient improves. It may be present in patients who are able to bend forward freely and without pain, while standing with the knees straight.

3. Pain and limitation of movement in the low back. This, too, varies with the severity of the symptoms. In some instances, the lumbar spine is flattened or even fixed in slight flexion by muscle spasm, all movements of the low back are markedly limited, and the patient may be unable to stand erect. In others, all movements are free and painless. Various gradations between the two extremes may be encountered in a series of patients, or in the same patient at different times. A common finding in a mild chronic case is that the patient is able to bend forward without difficulty, but has pain when straightening up.

In the more severe cases with sciatic radiation, pain may be elicited by pressure over the superior gluteal, sciatic, popliteal, or posterior tibial nerves, and the ankle jerk on the affected side may be diminished or absent. Sensory disturbances, muscle atrophy, and weakness have not been noted in this group of patients. When there is marked limitation of straight-leg raising and forward bending, the patient may have a peculiar stalking gait with the lumbar spine flattened; the lumbar spine is bent backward with the pelvis as the patient steps forward and straightens the knee. This condition may be present in patients who have no pain while walking.

The roentgenograms of the lumbar spine reveal nothing abnormal unless congenital anomalies are present, and these are not believed to be factors in the production of low-

back pain or sciatic pain. However, roentgenograms should be taken in order to rule out destructive disease (tuberculosis or neoplasms) or spondylolisthesis. The routine laboratory examinations show nothing abnormal. Lumbar punctures and spinograms have not been done on any of the author's patients who were under the age of nineteen years.

In the four patients whom we have operated upon, the fifth lumbar disc was involved. The lesion was not a rupture with extrusion of a sequestrum, but was a rather soft, elastic, domelike bulging of the annulus into the spinal canal. It has been our experience that, in adults, this type of lesion of the fifth lumbar disc often fails to show abnormalities in the myelogram. Consequently, we would not consider a negative myelogram as very important evidence that the patient was not suffering from an intervertebral-disc lesion.

PATHOLOGICAL FINDINGS

As far as we have been able to determine, the pathological findings of these disc lesions in children are different from those usually seen in adults. In the four patients operated upon, the annulus was intact, but presented a well-marked, domelike swelling which was elastic and fairly soft. When this was incised, semi-fluid grumous material exuded into the spinal canal. This was more liquid than is the usual nucleus pulposus material in adults. More of the same material was removed with a curette. In none of these cases were there any sequestra, or partially free masses of living or necrotic cartilage or fibrous tissue.

The microscopic examination of the material revealed normal intervertebral-disc tissue in three cases and this, plus some degenerating cartilage, in one case. These findings of the pathologist are what one would expect from the appearance of the material removed at operation.

PROGNOSIS

The prognosis varies with the severity of the symptoms and is usually good because, in most instances, the symptoms are relatively mild; the patients recover after conservative treatment and are able to resume their normal activities. It is unusual for one of these patients to have to stop school, even for a short time, on account of the back pain, as bed rest is rarely necessary. In the author's experience, patients whose symptoms have been so severe that they required prolonged bed rest have not been relieved except by operation. This has occurred in only about 10 per cent. of the patients with juvenile disc lesions whom we have seen, and in these four the symptoms were relieved by the operation.

From our observations, it seems probable that the thin mucoid material of the nucleus pulposus may become inspissated, permitting the disc to shrink, and thus the protrusion into the vertebral canal is lessened. It is also probable that the posterior portion of the annulus remains permanently thin and a point of lessened resistance; it may give way later and cause symptoms in adult life. On the other hand, it is possible that some of the narrowed discs, seen in adults who give no history of preceding low-back pain or sciatica, may be inspissated discs which were injured during childhood or adolescence. At any rate, the great majority of the disc lesions which occur in early life heal, either spontaneously or after conservative treatment, to a point where they no longer cause symptoms.

TREATMENT

As has been noted[1], the conservative treatment of intervertebral-disc lesions in adults varies with the severity of the symptoms. Theoretically, the ideal treatment would be to reduce the internal pressure in the bulging disc by putting the patient to bed and applying traction. Fortunately, this is not necessary in the average case and, in the author's experience, it does not promote cure in the severe cases. Consequently, most of these young people are treated on an ambulatory basis, and they are permitted to attend school. The treatment may be considered under the following seven headings:

1. *Rest:* The patient is placed on a regimen of relative rest, and is directed to avoid straining his back or even movements which aggravate the pain. Competitive athletics are forbidden, and he is excused from his gymnasium classes.

2. *Support:* A suitable support for the low back is prescribed in most instances. It not only protects the back from strain and lessens the pain, but it reminds the patient that his back is not normal and that it should not be subjected to any unnecessary exertion. The boys are fitted with a wide lumbosacral belt which extends from the lower three ribs to the trochanters. It is made of heavy canvas, its solid back is reinforced with stays, and it can be strapped or laced tightly and molded to the torso.

The girls are fitted with a laced girdle or corset, about as large as the lumbosacral belt, but made of lighter material. It is reinforced with stays in the back; the back should be solid and the laces should be placed in front or at one or both sides. This is not an elastic girdle, but one which gives a good deal of support to the back. It should be worn until the symptoms have subsided and the lumbosacral tenderness has disappeared, which may be several months.

If the patient is having considerable pain when first examined and no removable support is immediately available, the back may be strapped with adhesive, applied crosswise from one mid-axillary line to the other. The patient lies prone with a pillow under the abdomen; the shingle strapping is started at the level of the trochanters and is extended upward to grasp the lower three ribs.

3. *A Firm, Level Bed:* Even though the patient is comfortable in bed at night, he is directed to sleep on a firm mattress with a fracture board between the spring and the mattress.

4. *Postural Exercises:* After the limitation of straight-leg raising and of forward bending, and the local tenderness on deep pressure have disappeared, the patient is given postural exercises in order to strengthen the muscles of the abdomen and back.

5. *Manipulation:* Occasionally, the back is manipulated gently. The patient lies on the less painful side, facing the surgeon, with the painful limb hanging over the edge of the table. The surgeon grasps the patient's shoulder with one hand and pushes it backward and upward while, with the other hand or with the forearm, he suddenly pushes the ilium downward and forward, the dependent extremity aiding in this manoeuvre. The patient should be relaxed during this procedure. The manipulation may produce an audible crunch which may startle the patient, but rarely causes much pain. He should be warned of this before the manipulation is begun.

As stated previously, not much force is used and the manipulation is not repeated often. The author has removed ruptured intervertebral discs from a considerable number of adult patients who had had manipulations by osteopaths or chiropractors. In some of these patients the manipulative treatment had at first resulted in temporary relief, but the symptoms had recurred with increased severity; in others, relatively mild symptoms became so severe after the manipulation that operation was indicated for relief.

In young people, disc lesions do not seem to be the result of degeneration, and there is no displaced sequestrum which can be reduced with relief of symptoms. Consequently, manipulation would not be expected to be a very effective form of treatment in young people, and this has been the author's experience. Time and relative rest are the important factors in the healing of disc lesions in the young, just as in adults.

6. *Medication:* Little medicine is used. Occasionally, acetylsalicylic acid or codeine is given for pain, when this seems indicated. Vitamin B in relatively large doses is given to all patients with chronic low-back pain, because it seems to give them a feeling of well-being.

7. *Operation:* In about 10 per cent. of the cases, the symptoms are severe and disabling and do not yield to conservative treatment. These patients should be operated upon and the offending disc should be removed.

SUMMARY

Lesions of the lumbar intervertebral discs may occur in older children and in adolescents; the clinical picture differs from that usually seen in adults. If one admits that the patients with relatively mild symptoms are suffering from intervertebral-disc lesions, then the condition is common in early life, but it is rarely diagnosed. The typical disc syndrome may also occur in early life, but is rare. The prognosis is good, and nearly all of the patients recover as a result of conservative treatment.

REFERENCES

1. KEY, J. A.: The Conservative and Operative Treatment of Lesions of the Intervertebral Discs in the Low Back. Surgery, **17**: 291–303, 1945.
2. KEY, J. A.: Intervertebral Disk Lesions Are the Most Common Cause of Low Back Pain with or without Sciatica. Ann. Surg., **121**: 534–539, 1945.
3. KEY, J. A.: Idiopathic Low Back Pain and Sciatica—25 Years Ago and Now. Bull. Johns Hopkins Hosp., **80**: 217–230, 1947.
4. LOVE, J. G.: The Disc Factor in Low-Back Pain with or without Sciatica. J. Bone and Joint Surg., **29**: 438–447, Apr. 1947.
5. WAHREN, HERMAN: Herniated Nucleus Pulposus in a Child of Twelve Years. Acta Orthop. Scandinavica, **16**: 40–42, 1945.

DISCUSSION

POTT'S PARAPLEGIA

(*Continued from page 96*)

was 4.8; while, in the paralyzed group, the disease was more extensive and it involved 7.5 vertebrae. Therefore, patients with involvement of the mid-thoracic region are more likely to have paralysis; the more extensive the lesion, the greater the chance for paralysis.

From a study of these cases, it is evident that the paralysis is not caused by stretching of the cord over a sharp angle of bone, because many of the non-paralyzed patients showed a greater kyphos than did those who were paralyzed. The paralysis in the early cases seems to be caused by the pressure from the products of inflammation. Therefore, when the tuberculosis subsides with rest and other treatment, the pressure becomes less and the patient regains the use of his lower extremities.

Only three of the twenty patients were treated by laminectomy. In these, five and eight months, respectively, were required for the paralysis to clear. One showed no improvement. In the seventeen treated without operation, inflammation subsided in 3.8 months on the average.

Fifteen patients, whose paralysis developed early in the course of the tuberculous disease, made a complete recovery. In these cases, the paralysis occurred within the first year of the tuberculosis; it is believed that it was due to external pressure on the cord. Those patients whose paralysis developed five years or more after the onset of the tuberculosis did not improve; the cord was probably damaged by invasion of tuberculosis.

I agree with the teachings of Hibbs and others that early spine fusion is the best treatment for tuberculosis of the spine. Some years ago, I reported fifty cases treated by fusion and compared these with fifty treated without fusion. Every patient who had had adequate fusion had a good result. Since early fusion is now being done in tuberculous spines, we are not seeing the complication of paraplegia. I have not had a patient with paralysis in the last ten years. Prevention of paraplegia by early spine fusion is the treatment of choice.

DR. GEORGE J. GARCEAU (closing): Dr. Cleveland and Dr. Kite have stated that early spine-fusion operation and rest will prevent paraplegia. Almost all of our patients had been treated by bed rest for a long period. Eleven of the thirty-two patients had had spine-fusion operations before the onset of the paraplegia. In only one of these patients was an unsound fusion found at the time of operation for relief of the paraplegia. Dr. Cleveland has just said that eighteen of thirty-one patients in his series also had had spine-fusion operations prior to the onset of the paraplegia. His experience is comparable to ours.

Dr. Kite stated: ". . . the paralysis is not caused by stretching of the cord over a sharp angle of bone". This is rarely observed, but it does occur over a sequestrum and in pathological dislocation of the spine, as seen in Figures 1-A and 2-A of our paper.

I would like to paraphrase Dr. Cleveland's quotation from Percival Pott's paper: What we have affirmed is what we have seen and what we believe we have proved, and the objections are merely speculative and theoretical.

STREPTOMYCIN IN TUBERCULOUS BONE AND JOINT LESIONS WITH MIXED INFECTION AND SINUSES *

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This clinical study has no relationship to the bacteriological investigation of streptomycin, the experimental study of its action, or the abstruse problems associated with its discovery and development, by means of animal experimentation, until its final use on the human. Since patients with bone or joint tuberculosis in whom sinuses have developed represent the greatest test for any form of treatment, and since the continuance or closure of a sinus is a clinical factor that can be ascertained with certainty, the primary part of this report is limited to such cases.

Patients With Sinuses

The series to be reported includes ninety-five patients who had bone or joint tuberculous lesions with mixed infection and sinuses. These patients had thirty-nine additional bone or joint foci without sinuses. The ninety-five patients had 107 separate foci with sinuses, several of them having more than one tuberculous lesion of the bones and joints with mixed infection. There were 172 separate sinus tracts, and these had innumerable openings to the surface.

All records were carefully analyzed statistically. No essential change in response to treatment with streptomycin could be attributed to race, color, sex, or occupation. Age apparently did not affect the speed of healing of sinuses in children; they did, however, tolerate the drug better than adults. No complications occurred among the children, except that one patient who had meningitis became deaf. However, loss of auditory function may result from meningitis, regardless of treatment.

Lesions with sinuses were located as follows: fifty-three in the spine; ten each in the hip and knee; seven in the sacro-iliac joint; four each in the wrist, elbow, sternum, and costal cartilage; three in the rib; three in the clavicle; and one each in the scapula, mandible, metatarsal, metacarpal, and trochanteric bursa. In the main, the location of the primary tuberculous orthopaedic focus had no relationship to healing or to action of the drug. There was one exception to this,—namely, the sacro-iliac joint. Of the seven sacro-iliac lesions, three healed, three did not heal, and one patient died of meningitis while the local lesion was healing rapidly. The observation has been made at Sea View Hospital for many years that sacro-iliac tuberculous lesions were potentially more serious than similar infections in other bones or joints.

Bacteriological Findings

The ninety-five patients studied all had proved tuberculous organisms in their lesions. No effort was made to determine whether the organism was human or bovine, or whether it was resistant to streptomycin. It soon became apparent that the type of mixed-infection organism or secondary invader was not important as regards the reaction to the drug. The secondary invaders included *Escherichia coli*, *Staphylococcus albus*, *Staphylococcus aureus*, streptococci, gram-negative bacilli, and diphtheroids. Anaerobic organisms were not cultured. No specific type of bacteria in the healed or unhealed series was found in greater incidence than any other organism.

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 25, 1949.

In only one patient to date has the sinus reopened. This patient's original mixed-infection organism was a diphtheroid, but the reopened sinus was sterile to culture. In this case elbow resection was done, followed by primary healing. The wound has remained healed.

Penicillin had been used in many of these cases before streptomycin was available. Tubercle bacilli could routinely be cultured following the use of penicillin alone. Almost immediately after the institution of streptomycin therapy (within a period as short as three days), acid-fast organisms usually were unobtainable by culture. In one case, however, they persisted for ten weeks, until the patient's death from meningitis. It is the authors' feeling that it is hard to obtain a positive culture after streptomycin therapy has been started. Frequently cultures became sterile for all other organisms following the use of streptomycin, although the sinuses continued to drain. The combined use of penicillin and streptomycin produced no advantage, either in the culture or in the clinical picture.

TREATMENT

Method and Dosage

Early in the study 2 grams of streptomycin a day, in divided doses, was given every six hours for the first week; thereafter 1 gram a day was administered. After one-third of the patients had been treated, the amount was changed to 1 gram a day, and this was continued throughout the course. Where necessary because of complications or other factors, the dosage was reduced to 0.5 gram a day. When approximately half of the series had been completed, the dosage of 1 gram a day was divided so that 0.5 gram was given twice a day, and that is the authors' custom at present. No noticeable change in response occurred, regardless of whether 1 gram or 0.5 gram a day was used. However, the patients who received 0.5 gram a day were susceptible to the streptomycin, and perhaps its effectiveness may be increased by personal susceptibility. Early in the study, when streptomycin was scarce, the patients were maintained on the drug for only a few days after healing had occurred. Occasionally it was impossible to continue the drug for a satisfactory period because of its expense and scarcity. Improvement would be noted, but because the sinus persisted, the patient would again have to be placed on the drug to secure complete healing.

The time required for healing seemed to be definitely related to the period of intake of the drug, rather than to any continued effect of streptomycin once its administration had been stopped. For instance, one patient was given streptomycin at irregular intervals for one year. Healing occurred when he had been treated for a total of 103 days. Another patient received the drug sporadically over a period of eight months, but healing did not take place until treatment had been given for 108 days. In another patient, given the drug for 110 consecutive days, healing occurred at 106 days.

In some cases in which streptomycin could not be continued until healing had occurred, the initial treatment produced such good general physical response that the sinus subsequently healed without further medication. This was not a common finding.

The greatest amount of streptomycin used in one case was 697 grams, given to a fifty-five-pound child (51 grams were administered intrathecally). In this patient, with proved tuberculosis of the hip and proved tuberculous meningitis, there was complete arrest of both the meningitis and the hip lesion. The scarred hip was subsequently arthrodesed. The microscopic examination of tissue removed at the time of arthrodesis was negative for tuberculosis.

The smallest amount of streptomycin used was 21 grams, given to an adult with tuberculosis of the cervical spine, whose sinuses had drained for two years. Healing occurred in ten days and the sinuses have remained healed. This patient had had a spine fusion of adequate extent and continuity before the streptomycin was given, but his sinuses had not been prevented or healed.

The shortest time required for healing was seven days in a patient who had had a

sinus for four months over an area of rib destruction, associated with tuberculosis of the thoracic spine. Spine fusion was carried out successfully following healing of the sinus, 98 grams of streptomycin being given during a period of ninety-one days. This patient's sinus has remained healed to the present.

The average healing time showed no definite relationship to the amount of drug used. Where the patient tolerated only 0.5 gram a day, healing occurred at the same relative speed as where a full gram a day was tolerated. It may be that the patient's tolerance to the drug is related to the speed of healing. We are not suggesting that the amount of drug routinely given be less than one gram a day. We do suggest, however, that one need not force one gram a day if complicating circumstances indicate a reduction in the daily amount. The healing time is definitely related to the duration of treatment rather than the total dosage.

Duration of Treatment in Healed Cases

Fourteen patients were treated for less than 90 days, forty from 90 to 120 days, sixteen up to 200 days, and nine had more than 200 days of treatment. Nine months' treatment, or more, may be required to secure healing in some instances. On the average, healing occurred at about sixty days (60 grams of streptomycin), yet the treatment was carried on for an average of ninety days (90 grams). It might be suggested, therefore, that, to ensure an optimal chance of healing being maintained, the time required for healing be taken as a guide for the period of continuing the drug after healing. Streptomycin should be continued after sinus closure for a period equal to half the length of time necessary to secure healing. Therefore, if 100 days are required to secure healing, streptomycin should be given for another fifty days. This suggestion is presented for consideration and not as a directive, in view of the relatively short experience in the use of the drug.

RESULTS

Toxic Manifestations

Four main types of toxic manifestations were noted. These were referable to the auditory branch of the eighth cranial nerve, to the vestibular branch of the eighth cranial nerve, to the skin, and to allergic asthmatic reactions.

Deafness occurred once, but was disregarded in the treatment, since the child had proved meningitis. The deafness had been said to be complete, but the child now reacts to sound. There was no involvement of the vestibular branch of the eighth nerve in this patient.

The symptoms of headache, vertigo, tinnitus, dizziness, nausea, and vomiting occurred in varying degrees in fifteen patients. In all of these patients the drug was either maintained, or, after a short interval of withdrawal, its use was resumed. In five, satisfactory relief was secured by the administration of benadryl. In four cases, benadryl failed to improve the symptoms. No permanent damage occurred in any of these patients, and all were entirely relieved of symptoms following removal of the drug at the completion of treatment.

The skin symptoms consisted of severe itching in three patients and varying degrees of dermatitis in three others. One of these patients, although suffering from severe dermatitis, is still being maintained on the drug, and the dermatitis is receding. The patients with pruritis all responded satisfactorily to benadryl, and the use of streptomycin was not interrupted. Where actual dermatitis existed, benadryl failed to relieve the situation. In all of the dermatitis cases, the treatment was either carried on with reduced dosage or, after a short interval of rest, streptomycin was resumed at reduced dosage and the treatment was continued. Following completion of treatment with streptomycin, the dermatitis completely cleared in two cases; the third patient has already been mentioned.

Moderately severe asthmatic symptoms, necessitating oxygen therapy for ten days

during use of the drug, were encountered in one case. This reaction subsided when the dosage of streptomycin was reduced to 0.5 gram per day. In this patient, benadryl failed to relieve the asthmatic symptoms.

Thus complications of some nature occurred in 24.2 per cent. of the cases in which streptomycin was used. In no instance was it necessary to stop the use of the drug permanently because of them.

Death During Period of Treatment

These patients were, on the whole, extremely sick; four died during the period of treatment. Two deaths were due to tuberculous meningitis, one to a cerebral accident, and one to kidney failure with severe amyloidosis. The one patient with proved meningitis recovered. No deaths were in any way related to the streptomycin therapy.

Benefits Derived from Streptomycin

The healing of sinuses, while dramatic, was not the only benefit directly attributable to the use of the drug. There was marked improvement in the general physical condition of the patients, as well as gain in weight. These were progressive changes which began within a few days following use of the drug and continued even after its removal; the peak of improvement occurred about six weeks after treatment was begun.

In eight of the patients, surgery would have been impossible without streptomycin. In five of these the disposition of sinuses preventedad equate surgery; and in three, emaciation was so severe that the patient's condition appeared hopeless at the time the drug was started. In eight of these patients the sinuses have healed; adequate surgery has been performed; and all are in a favorable condition for eventual recovery and discharge. In the five in whom sinus areas prevented operative care, incisional lines were made through the healed sinus areas; all of these incisions are at present healed. Three of the five patients had tuberculosis of the spine with disease of the posterior elements, and fusion was carried out through the healed scarred sinus area on the diseased posterior elements. The fusions are still secure and healing of the operative incisional areas has been maintained.

Failure of Healing of Sinuses

In sixteen of the ninety-five patients (16.8 per cent.), the sinuses failed to heal. Failure of healing was apparently due to the presence of a sequestrum, a thick-walled abscess, an uncollapsed bony pocket, or inadequate stabilizing surgery of the infected joint. To these causes of failure may be added an insufficient period of administration of streptomycin, or an insufficient life span of the patient to permit satisfactory drug administration.

No failures were seen which could not be explained satisfactorily by the factors just mentioned. Clinical failures due to the development of resistance of the tuberculous organism to streptomycin, we believe, require more proof. When a patient fails to respond, we review the case intensively, keeping in mind the factors mentioned. In one patient, seen recently, streptomycin has been continued in spite of allergic manifestations of asthma and exfoliative dermatitis, and without satisfactory response as regards the sinus. Intensive review of the roentgenograms of her spine has disclosed pseudarthrosis, not known to exist during the past five years. The pseudarthrosis had existed without the occurrence of further collapse of the vertebral bodies, but is causing continuance of manifestations through sinus drainage. In another patient with wrist-joint disease, in whom treatment failed, wrist resection disclosed an unsuspected carpal sequestrum.

Successful Healing of Sinuses

In seventy-nine patients (83.2 per cent.) the sinuses healed; all except one have remained healed. In one patient, with elbow-joint tuberculosis, the sinus reopened after

forty-two days, 49 grams of streptomycin being given. Cultures from the reopened sinus were sterile. Streptomycin was continued and elbow-joint resection was done, with primary healing of the incisional wound. The sinus has remained closed, although typical hypermobility of the elbow followed resection. To date, a total of 217 grams of streptomycin have been administered. The patient had other lesions involving the cervical spine, sternum, ankle, thoracic spine, lumbar spine, and left sacro-iliac joint, but the chest was negative. From being practically moribund, he has improved so that his general condition is excellent; his other tuberculous lesions are being stabilized surgically.

An instance of the efficacy of the drug alone is shown by a patient who had had a sinus draining from the sternum for thirty-five years. Sinus healing occurred in seventy days with 77 grams of streptomycin, without surgery or other treatment. This patient's treatment was continued, on the basis of clinical judgment, for a total of 105 days (112 grams of streptomycin). Her sinus has remained healed to the present. Even before this study was completed, on the basis of clinical judgment alone and without any definite formula for continuance of streptomycin, this patient's treatment was continued beyond the period of healing by 50 per cent. of the time necessary to secure healing. This case corroborates the authors' rule of continuing treatment, suggested previously.

SURGERY

As mentioned, streptomycin makes surgery possible in certain instances. It is evident that in all of the patients with sinuses in the present series, a certain amount of decompression of their abscesses exists. Frequently this decompression of abscesses and their drainage is not satisfactory. Surgical eradication of extremely thick-walled abscesses, especially about the spine, is, of course, not always possible. Excisional surgery of abscesses and granulation tissue at the time of arthrodesis of other joints is possible and important. Sequestrectomy is essential, where it can be accomplished. Arthrodesis of weight-bearing joints is essential if healing is to be secured; one cannot rely on streptomycin alone. Resection of non-weight-bearing joints is permissible. Surgery has accompanied the use of streptomycin throughout this study.

EFFECT OF STREPTOMYCIN IN TUBERCULOUS BONE AND JOINT LESIONS WITHOUT SINUSES

Although this investigation concerns tuberculous bone and joint lesions with sinuses, one cannot completely disregard concomitant lesions, present among those patients not having sinuses, which are affected by the treatment. Too short a time has elapsed for a complete study of this material, as changes are slow, viable organisms may persist for some time, and reactivation may occur at a much later date. Clinically, however, retardation of the progress of the unopened lesion and improvement of general health have been noted during the period of administration of streptomycin, on about the same basis as for the series of sinus-complicated cases. The authors are skeptical as to the ability of streptomycin alone to heal a bone or joint lesion permanently, even though a sinus is not present.

Five children with proved bone or joint tuberculosis, without sinuses, have recently been studied intensively at the House of St. Giles the Cripple. Two had knee infections; the others had infection of the tibia, hip, and spine. Four received streptomycin for ninety days (90 grams of streptomycin), with apparent temporary arrest of the condition, clinically and roentgenographically. Subsequently all four have been found to have active disease extension, either alone or accompanied by active pathological tissue and culture changes. In the girl who received 697 grams of streptomycin, proved residual disease activity did not occur. In other words, all the patients given 90 grams of streptomycin for ninety days continued to have active disease, despite what would in the past have been considered satisfactory streptomycin therapy. In only one child, given a tremendous amount, did the disease remain inactive. In all but one of the patients, this continued disease

activity was not suspected clinically. The question arises of whether, in closed bone and joint tuberculous lesions, one can entirely rid the body of the active tuberculous organism and its destructive process by the use of this drug alone. Apparently one cannot. Surgery still is a necessary adjunct.

SPECULATION

As we see the dreadful end results of this disease, we would raise three questions: First, do the complications involved in any way detract from the forceful use of streptomycin? We believe not.

Second, should we reverse all policy of the past and decompress closed abscesses by actual incisional drainage, administration of streptomycin, and subsequent surgery? We suggest that we should.

Third, can prolonged use of streptomycin eventually rid the body of disease and eliminate the necessity of surgical ankylosis of weight-bearing joints? With our present knowledge, it appears that it cannot. Control of a closed or open lesion with a reasonable amount of streptomycin, we believe, requires surgery. In most instances, by the time the disease process has been recognized and controlled, the involved joints are deformed and their sacrifice is justified in view of the protection afforded.

CONCLUSIONS

1. At the present time, streptomycin represents a tremendous advance in the treatment of patients with these lesions. Streptomycin undoubtedly will be improved upon and superseded by some other agent in the future, giving us better control of this disease and possibly enabling us to eradicate it.

2. In closed lesions streptomycin, in dosages of 90 grams in a period of ninety days, has failed to arrest the tuberculous process permanently.

3. Failure of treatment with streptomycin suggests strongly the presence of a sequestrum, a thick-walled abscess, or inadequate surgery.

4. Healing seems to be related to the duration of administration of streptomycin and not to the total dosage.

5. In patients having sinuses, streptomycin should be continued after healing for a period at least half as long as the healing period itself.

6. Surgical ankylosis, or resection of non-weight-bearing joints, is apparently as necessary now as it ever has been, but is much safer and more effective. Indeed, at times surgical attack is made possible only by the use of streptomycin.

NEUROPATHY OF THE MEDIAN NERVE DUE TO COMPRESSION BENEATH THE TRANSVERSE CARPAL LIGAMENT *

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Neuropathy due to compression of the median nerve at the wrist occasionally may be associated with advanced hypertrophic arthritis or with trauma involving the wrist joint or the carpus. In some of these cases the onset of the neuropathy may occur several years after injury, producing a so-called "tardy median palsy" which is, in most respects, similar to a "tardy ulnar palsy" as far as its pathogenesis is concerned. It is easy to see how a "tardy median palsy" might develop. The median nerve lies superficial to the flexor tendons of the fingers, immediately beneath the transverse carpal ligament **; these structures completely fill the carpal tunnel. Narrowing of the carpal tunnel secondary to fracture or hypertrophic arthritis may compress the median nerve between the bones of the carpus and the transverse carpal ligament. Zachary recently reported two such cases, one with old bilateral fracture of the carpal navicular and the other with a malunited Colles's fracture.

We are concerned here, however, with the spontaneous development of neuropathy due to compression of the median nerve beneath the transverse carpal ligament, without antecedent or associated injury or disease. Marie and Foix, in 1913, reported a case with severe bilateral atrophy of the thenar muscles; at autopsy, a definite neuroma of the median nerve was found, just proximal to the transverse carpal ligament. These authors felt that there had been a so-called strangulation of the nerve beneath the ligament and they suggested that, if the diagnosis could be made early enough in other similar cases, section of the ligament might prevent atrophy of the thenar muscles. Moersch, in 1938, reported a case of median neuritis with bilateral atrophy of the thenar muscles. Although no treatment was instituted in this case, Moersch also suggested that the transverse carpal ligament be sectioned early, to prevent irreversible changes in the median nerve.

In 1946, Cannon and Love reported a series of thirty-eight cases of "tardy median palsy". In nine of these cases, surgical division of the transverse carpal ligament was carried out with satisfactory results. Only three of the nine cases, however, apparently can be classified as spontaneous median palsy, without associated injury or disease. Brain, Wright, and Wilkinson presented, in 1947, a detailed report of six cases of spontaneous bilateral compression of the median nerves in the carpal tunnel. All six cases were treated successfully by section of the transverse carpal ligament. These workers also stated that they had seen eight additional patients in whom the diagnosis had not been verified by surgical exploration.

In the past year the authors have observed four cases of spontaneous neuropathy, due to compression of the median nerve beneath the transverse carpal ligament. Three of these cases, reported here, have been treated by surgical division of the transverse carpal ligament, with excellent results.

CASE 1. A negro woman, thirty-four years old, was first seen on January 15, 1948. She complained of periodic episodes of numbness in both hands, lasting from a few minutes to half an hour. These symptoms had been present for a year in the right hand and for six or seven months in the left hand; they were much less severe in the left hand. The numbness and paraesthesia were always limited to the thumb, the index finger, and the middle finger; and were aggravated by carrying heavy objects or by active use of the hands.

* Read at the Annual Meeting of the American Society for Surgery of the Hand, Chicago, Illinois, January 21, 1949.

** Some anatomists call this structure the volar carpal ligament.

The paraesthesia was always worse at night. The patient had been aware of increasing difficulty in manipulating objects with her right hand. She worked as a cook and was unable to peel more than one or two potatoes continuously without stopping, because of numbness and tingling in her right hand. There was no history of injury to her hands or wrists.

Physical examination disclosed no limitation of motion in the cervical spine, no tenderness over the scaleni, no pain on jugular compression, and no alteration in the tendon reflexes. The Tinel sign was positive over the median nerve at the wrist, bilaterally, but there was no evidence of thenar-muscle atrophy or weakness of any intrinsic muscles in either hand. On the right hand, anaesthesia was present over the volar aspect of the thumb, index finger, middle finger, and radial half of the ring finger; and hypoaesthesia over the medial half of the palm and over the dorsal aspect of the distal two phalanges of the thumb, index finger, and middle finger. On the left hand there was also hypoaesthesia in the median distribution, but this was much less severe than on the right.

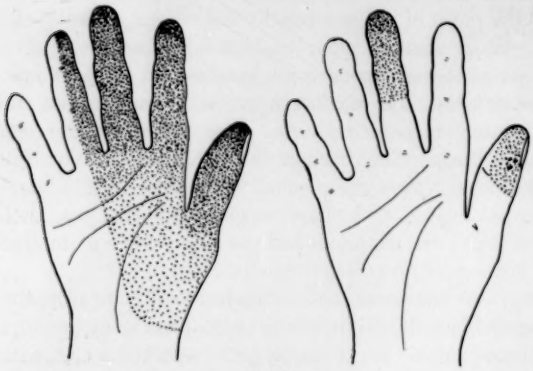

FIG. 1

Case 1. Diagram of volar aspect of the right hand, showing extent of anaesthesia prior to section of transverse carpal ligament on March 10, 1948, and that remaining four and one-half months later.

On March 10, the right median nerve was explored and the transverse carpal ligament was sectioned. The nerve was found to be somewhat gray in color; it was slightly enlarged, proximal to the carpal ligament, but there was no evidence of a true neuroma. The connective tissue immediately adjacent to the nerve appeared to be a little more adherent than normal. The sheath of the nerve was distended with normal saline to exclude the possibility of an interstitial neuroma.

Immediately after recovery from the anaesthesia, the patient stated that she no longer had pain or tingling in the fingers. During the next six months, sensation in the right hand gradually returned (Fig. 1). When the patient was examined on July 22, there was persistent hypoaesthesia over the radial aspect of the distal phalanx of the thumb and the entire volar aspect of the distal two phalanges of the middle finger. By January 15, 1949, normal sensation had returned to the entire hand, except for the persistence of slight hypoaesthesia over the volar aspect of the distal phalanx of the middle finger.

This patient is now able to work hard with her right hand without any discomfort or weakness. The numbness in the left hand has remained unchanged; but, because it is causing so little disability, operation on the left wrist was not advised at this time. The patient was warned, however, that an operation should not be delayed if there was any increase in the degree of numbness in her left hand.

CASE 2. A fifty-year-old Jewish woman was first seen at the Clinic on November 22, 1947. She complained of pain and hyperaesthesia in the thumb, index finger, middle finger, and ring finger of each hand, of eight months' duration. The condition was worse in the right hand than in the left. She was unable to do a great deal of her housework, because it aggravated the pain in her fingers. The pain frequently awakened her at night. Over the preceding few months she had found it increasingly difficult to manipulate small objects with her right hand. There was no history of injury to either wrist.

Physical examination revealed the presence of hyperaesthesia throughout the entire median distribution in both hands, more pronounced on the right. There was no evidence of muscle atrophy or weakness in either hand. Marked tenderness and slight thickening were observed over the sheath of the short extensor and long abductor tendons of the thumb in the region of the radial styloid on the right. The Tinel sign was positive over the median nerve at the right wrist.

A diagnosis was made of bilateral median neuritis and of stenosing tenosynovitis (de Quervain's disease) at the right wrist. A cervical sympathetic block was performed on the right, but no objective or subjective improvement in the patient's condition resulted.

The patient returned on January 13, 1948, and was first observed in the Department of Orthopaedic Surgery. She stated that the right thumb, index finger, and middle finger felt numb all of the time, although there was still severe hyperaesthesia in the median distribution of the hand. She was finding it progressively difficult to sew or do her housework. The grip in the right hand was weak because of pain in the region of the radial styloid. There was no evidence of atrophy or weakness of the thenar muscles.

The wrist and thumb were immobilized with a splint, which the patient was instructed to wear most of the time. Physical therapy, in the form of paraffin baths, was ordered, but this treatment was discontinued because the patient complained that the heat aggravated her pain.

Although somewhat improved three months later, she was still unable to work with her right hand. On April 22, the median nerve was explored, the transverse carpal ligament was sectioned, and the sheath of the long abductor and short extensor tendons to the thumb was incised. This tendon sheath was thickened and contained an increased amount of fluid. The median nerve appeared normal, with no demonstrable neuroma or compression.

About one week after the operation the patient admitted, for the first time, that there had been significant improvement in her right hand. The pain in her thumb was gone and there was less hyperaesthesia in the median distribution. Six weeks after the operation, she reported that she no longer had numbness or tingling in her fingers. Tinel's sign at the right wrist was absent. When last seen, almost nine months after operation, she stated that her right hand was entirely normal; there was no weakness of grip or sensory changes; and she was able to use the hand without discomfort. There was little change in the condition of the left hand, but the disability was too slight to justify operation on the left wrist.

CASE 3.-A white woman, forty-seven years old, was first seen at the Clinic on May 12, 1948. She stated that, in August 1947, she had first noticed pain in her left shoulder when she raised her arm. She consulted a local physician, who ordered roentgenograms of the cervical spine and diagnosed the ailment as a protruded cervical intervertebral disc. About one month later she noticed numbness in the tip of the right middle finger. A novocain injection of the right anterior scalenus afforded some relief from the shoulder pain, but did not affect the numbness in her right hand. This progressed slowly for a period of several months, to involve the thumb, index finger, middle finger, the radial side of the ring finger, and the median distribution in the palm. At the time of her admission to the Clinic, there was numbness in the median distribution in the right hand and a burning and tingling feeling over the median distribution in both hands. This burning increased at night and was aggravated when the patient did any type of work with her hands. In the preceding few months she had noted increasing weakness in the right hand and difficulty in manipulating small objects. There was no history of injury to either wrist or hand.

Physical examination showed severe hypoaesthesia over the entire median distribution in the right hand and over the distal phalanx of the left thumb. The Tinel sign was positive at both wrists over the median nerve. There was definite atrophy of the right thenar muscles, with weakness of the opponens and short flexor of the thumb. Tenderness was present over the mid-cervical region posteriorly, and neck movement was slightly restricted in all directions. There was no tenderness over the scaleni, however, and no evidence of a cervical rib.

Roentgenograms of both wrists disclosed nothing abnormal. Roentgenograms of the cervical spine showed narrowing of the fifth interspace with mild hypertrophic changes in this region.

On June 18, 1948, the right median nerve was explored and the transverse carpal ligament was sectioned. A fusiform swelling of the nerve was found, just proximal to the carpal ligament and extending for a distance of about two centimeters, which seemed a little more pronounced than the normal anatomical enlargement of the nerve in this area. The nerve appeared firmer in this region than elsewhere, but no definite neuroma could be demonstrated.

Immediately after the operation, the patient stated that she had been relieved of the burning sensation and pain in the fingers of the right hand; one month later there was a cessation of the burning and tingling in the hand. The Tinel sign was negative, although the grip was still weak. On October 25 (four months after operation) the patient stated that she no longer experienced pain in her right hand; there was considerable improvement in strength of grip. She could hold a knife, write letters, and do light housework without discomfort. Her paraesthesia had cleared completely, and the hypoaesthesia had improved.

On January 16, 1949, the patient returned because of progressive pain and hypoaesthesia in the median distribution of the left hand, and a "snapping tendon" in the middle finger of the right hand. Examination revealed improvement in the atrophy of the right thenar muscles; the grip was almost normal; and there were no longer sensory changes in the hand. A tender swelling could be palpated in the flexor tendon, overlying the head of the third metacarpal, and this tendon could be felt to snap beneath the annular band when the finger was passively extended. At times this finger would be locked in flexion and could be extended only passively. In the left hand there was moderate atrophy of the thenar muscles, with weakness of grip and numbness in the median distribution. The Tinel sign was positive over the median nerve at the left wrist.

On January 17, 1949, both hands were operated upon. The ligamentous sheath, or annular band, overlying the flexor tendons of the right middle finger was incised. The transverse carpal ligament of the left wrist was sectioned. No definite abnormality in the left median nerve could be demonstrated.

When the patient was last seen, on February 28, there were no complaints referable to the right hand. In the left hand, only slight hypoaesthesia remained over the two distal phalanges of the middle finger and the distal phalanx of the thumb. Strength of grip in the left hand had improved, and there was less atrophy of the thenar muscles.

DISCUSSION

These cases represent the spontaneous development of neuropathy, due to compression of the median nerve beneath the transverse carpal ligament. Surgical section of the

ligament evidently decompresses the nerve sufficiently to restore its normal function and to relieve the patient's symptoms. Although the signs of nerve compression were found not to be severe at the time of operation, release of the nerve by section of the transverse carpal ligament afforded prompt relief in each case.

The pathogenesis of this condition is not clear. Occupation may be a causal factor, since Brain and his co-workers have shown experimentally that there is a definite rise in pressure within the carpal tunnel when the wrist is strongly extended. Certainly active use of the hands aggravated the symptoms in each of the patients reported here; but none of them had been performing very strenuous work with the hands. It seems unlikely that occupation alone could be considered a causative factor, or the condition would be encountered much more frequently than it evidently has been.

Ischaemia of the nerve due to pressure may play some etiological role, especially since, in most of the cases reported, the patients have been past fifty years of age. Our first patient, however, was only thirty-four. Sex may have some relationship to the condition. The six cases reported by Brain and the authors' four cases were all in women.

The presence of de Quervain's disease in one of the authors' cases and of so-called "trigger finger" in another case might be an indication that trauma of a similar nature could produce the median neuropathy. The treatment of the two conditions—stenosing tenosynovitis and spontaneous median neuropathy—is certainly similar.

Of special interest is the fact that none of the patients has had any disability from section of the transverse carpal ligament. A little bulging of the flexor tendons at the wrist has been noted, especially when the wrist is flexed; but with the wrist in extension—which is the position of function—there is apparently no weakness in the power of flexion of the fingers.

Great care should be taken in making the diagnosis of spontaneous compression of the median nerve in the carpal tunnel. A complete neurological and orthopaedic examination must be made to rule out the presence of progressive muscular atrophy, syringomyelia, protrusion of a cervical intervertebral disc, or a scalenus anterior syndrome. The presence of a positive Tinel sign over the median nerve at the wrist, as well as the strict limitation of all sensory findings to the median distribution, distal to the wrist, are the two most reliable diagnostic findings. The condition is apparently always bilateral, but symptoms are usually more noticeable in the hand which the patient uses more. Roentgen-ray examination of the wrist is helpful in ruling out the presence of hypertrophic arthritis or bone injury that may be causing pressure on the median nerve.

The authors are confident that this condition is not a rarity, as one might assume from the paucity of previous reports in the literature. If this condition is kept in mind during the examination of patients who have symptoms of median neuritis, more cases are certain to be recognized.

REFERENCES

BRAIN, W. R.; WRIGHT, A. D.; and WILKINSON, MARCIA: Spontaneous Compression of Both Median Nerves in the Carpal Tunnel. Six Cases Treated Surgically. Lancet, **1**: 277–282, 1947.

CANNON, B. W., and LOVE, J. G.: Tardy Median Palsy; Median Neuritis; Median Thenar Neuritis Amenable to Surgery. Surgery, **20**: 210–216, 1946.

MARIE, PIERRE, et FOIX: Atrophie isolée de l'éminence thénar d'origine névritique. Rôle du ligament annulaire antérieur du carpe dans la pathogénie de la lésion. Rev. Neurol., **26**: 647–649, 1913.

MOERSCH, F. P.: Median Thenar Neuritis. Proc. Staff Meet., Mayo Clin., **13**: 220–222, 1938.

ZACHARY, R. B.: Thenar Palsy Due to Compression of Median Nerve in the Carpal Tunnel. Surg., Gynec., and Obstet., **81**: 213–217, 1945.

A METHOD OF MOBILIZING THE TEMPOROMANDIBULAR JOINT

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As man must chew in order to relish and assimilate solid food, inability to open one's mouth constitutes a serious handicap. Moreover, in the presence of ankylosis of the jaw, oral hygiene becomes difficult; sore throat cannot be attended; and infected tonsils must go without surgery. Dentition in children and decay of teeth in adults give rise to problems which cannot be coped with adequately unless the jaw is first mobilized. The conditions which cause partial or total immobility of the jaw may be grouped under five general headings: (1) congenital anomalies, such as syndesmosis or synosteosis of the mandible to the fixed bones of the skull; (2) injuries, including fractures of the mandibular condyle, old unreduced dislocations, depressed fractures of the zygomatic arch, gunshot wounds, and traumatic or degenerative arthritis; (3) inflammatory or rheumatoid arthritis; (4) pyogenic joint disease; and (5) cicatricial contractures of the soft tissues, such as follow burns, scalds, and suppurative infections of the walls of the mouth.

It is not necessary to discuss separately or in detail the different conditions which cause immobility of the jaw. With slight variations, the outcome in all is the same,—the mouth cannot be opened freely. Where there is solid bony fusion, the jaws cannot even be pried apart. When the immobility of the jaw is due solely to cicatricial contractures of the cheek, replacement of the scarred skin by pliable flaps will release the mandible. Occasionally, when immobility is the result of fibrous ankylosis, skeletal traction through the chin may stretch the adhesions in and around the temporomandibular joint, enabling the patient to open his mouth partially. In most instances, however, fibrous or osseous fusion of the mandible to any of the fixed bones of the skull points to one necessary solution,—surgical mobilization of the mandible.

HISTORICAL PERSPECTIVE

Resection of bones and their articular ends, which was in great vogue throughout the nineteenth century, must be regarded as the logical precursor of all arthroplasties. In the temporomandibular joint, where stability is not a factor, adaptive remodeling of the opposed ends of the bones is hardly warranted. All that is needed is to excise sufficient bone to create a space, and to resort to such contrivances as will prevent the freshly cut osseous surfaces from coming together and effecting reunion.

Although Valentine Mott, Lisfranc[17], Syme, and other workers had previously resected considerable portions of the mandible for tumors, Professor Humphry was the first, in 1856, to report excision of the condyle for relief of ankylosis of the jaw in arthritis. Humphry exposed the temporomandibular joint by a curved incision which began at the side of the orbit, ran transversely over the zygoma to the ear, and then took a sharp turn upward.

The European surgeons of the period were beset by the idea that in most instances immobility of the jaw was due to cicatricial contractures. Dieffenbach had advocated creation of pseudarthrosis in the ramus of the mandible. In line with this teaching, about 1855, Esmarch removed a wedge-shaped piece of bone from the body of the mandible through an incision in front of the masseter. Two years later, Rizzoli of Bologna recommended cutting the jaw bone with a powerful forceps, introduced by way of the mouth. Later Rizzoli advised the interposition of a piece of gutta-percha between the cut surfaces of the bones to prevent their reunion. Heath stated that in 1862, Clendon, a dental

surgeon in London, had devised shields to cap the upper and lower sets of teeth, and had inserted a wedge between them in order to keep the mouth open. Bottini, also of Bologna, found the use of a wedge very irksome to the patient and resorted to Humphry's original operation of condylectomy in a case of fibrous ankylosis of the temporomandibular joint. The operation failed when performed on one side; it was successful when the condyle on the opposite side was also resected. Bottini is said to have approached the temporomandibular joint through a vertical incision in front of the ear.

In the period which followed, and up to the present time, very little has been added to the pioneer work of Humphry, Esmarch, Rizzoli, Clendon, and Bottini. In 1875, Goodwillie of New York modified Clendon's shield and wedge and perfected a hinged, double-planed, oral speculum with a screw device which, when turned, forced the jaws to converge or diverge. Numerous varieties of mouth gags, and spring or elastic instruments have been devised to help the patient open and close his mouth and to exercise and strengthen the atrophic muscles of mastication.

The incisions employed by Humphry, Esmarch, and Bottini have been suggested time and again with but slight modifications and with claims of originality. Humphry's excision of the condyle was extended by Samuel D. Gross to include the coronoid process as well; Esmarch's resection of a wedge of bone in front of the masseter was transposed to the ramus behind it, even to the neck, below the condyle. In 1873, James Little of New York made an incision along the lower border of the masseter; he stripped the muscle until the neck and the condyle were exposed and resected a segment of the neck, leaving the fused head *in situ*. Little's operation was repeated by Robert Abbe of New York in 1880. Abbe approached the joint through a vertical incision in front of the ear; a horizontal incision, meeting its upper end, was carried along the lower border of the zygomatic arch.

Rizzoli's original idea of inserting gutta-percha between the cut ends of the bones has gone through successive modes of interposing muscle and fibrous tissue [14], a flap from the masseter [21], a pedicled flap of fascia, fat, and capsule [23], free fat and fascia [16], a cartilage graft [24], tantalum foil [7], and finally oxycel gauze. Toward the end of the last century and earlier in the present century, arthroplasty was practised in the true sense of the word: The cut ends of the bones were reamed, reshaped, and fitted together; and the raw surfaces were covered with elaborately designed fascial flaps which were sutured over and around the bones. In an attempt to gain greater exposure, as was required by all of these complicated procedures, the facial nerve or its branches were cut more than once; the internal maxillary artery was punctured while the fascial flap was being tacked around the newly carved condyle; the already attenuated roof of the articular socket of the temporal bone was deepened by a motor burr at the great risk of boring a hole into the middle cranial fossa; often the anterior wall of the external auditory meatus was torn,—an accident which was frequently followed by infection and putrid sloughing of the fascial flap through the rent in the ear.

For a joint in which the primary function is movement and not stability, attempts at adaptive remodeling and relining of the articular ends of the bones do not represent the sound practice of surgery. The much simpler bone resection, practised almost a century ago, requires less time, less room, and less tugging and tearing than arthroplasty; it involves less hazard. Mere resection of the articular ends is as effective as arthroplasty, if enough bone is excised and the severed ends are kept apart until the muscles have regained their functional usefulness.

BASIC PROBLEMS

Fixation of the Mandible

With the exception of tympanic ossicles, the mandible is the only bone of the skull which is capable of independent movement; all the rest, fused together firmly, move as a whole. Movements of the jaw at the temporomandibular joint are compromised when

the mandible becomes bound to the fixed bones of the skull by either fibrous bands or osseous bridges. Fixation of the mandible to the temporal, the zygomatic, or the maxillary bone necessarily checks its movements at the temporomandibular joint. When one holds the jaw still and tilts the head backward, the mouth opens, due to the movement of the skull at the temporomandibular and atlanto-occipital joints away from the fixed mandible. The supplementary motion of the fixed bones of the skull backward at the atlanto-occipital joint increases the range to which the mouth can be opened by the depression of the jaw. When the mandible is fused to the fixed bones of the skull, however, it moves with the calvarium at the atlanto-occipital joint and the mouth fails to open. Moreover, when the temporomandibular joint is fused or interlocked on one side, the articulation on the other side fails to permit motion. Frequently, although the temporomandibular joint on one side may not have been involved in the primary disease, it undergoes secondary degenerative changes in the course of prolonged immobility caused by the primary disease on the opposite side. There are instances, however, as in Marie-Strümpell type of inflammatory arthritis, when both joints are primarily, although not simultaneously, affected.

Changes in the Muscles

Irrespective of whether the ankylosis of the temporomandibular joint is unilateral or bilateral, when it has existed for a protracted period of time, the muscles of mastication undergo fibrotic changes and become shortened. They may never recover their contractility, even after wide resection of bone. This is especially true in adults, particularly in Marie-Strümpell type of inflammatory arthritis. In children with congenital fusion between the mandible and the maxilla, the temporal, the pterygoid, and the masseter muscles on the affected side may be completely wanting or fibrotic. However, as long as there are muscles on the opposite side, they can be helped to recover their contractile strength postoperatively by judicious stretching and exercise against tension.

Growth Arrest of the Mandible and Asymmetry of the Face

The mandible forms the skeletal framework of the lower portion of the face. In ankylosis of the temporomandibular articulation, there is often considerable facial asymmetry, especially when the fixation is unilateral and starts early in life. The body of the mandible is laid in halves, which fuse at the symphysis in the second year. At birth, the right or left ramus is represented by an articular condyle which is flush with the alveolar rim, and a coronoid process which rises slightly above as a small triangular bud. The mandible attains its adult form and configuration by an incessant process of removal and replacement of bone and, although it gains in bulk and alters somewhat in shape, its relation to the neighboring structures, especially to the maxilla, remains unchanged. With greater demand on mastication and with dilation of the paranasal sinuses, the maxilla expands forward, downward, and sideways. To keep pace with the three-dimensional expansion of the bone above, the body of the mandible pushes its way ahead, descends, and spreads its limbs apart. The ligaments and the muscles, which moor the rami to the fixed bones of the skull, resist this shift. In consequence, each ramus stretches to a considerable height, inclines forward, and twists inward around its long axis.

The question as to whether the rami acquire their height mainly by enchondral ossification, brought about by the proliferation of the articular cartilage which caps the condyles, or by extension of the membranous bone from below, is still debatable. In ankylosis of the temporomandibular joint, crossing of the cartilage plate by fibrous strands or bony trabeculae may in part be responsible for the arrest of growth and stunting of the ramus or of the mandible as a whole. Perhaps more important is the fact that fixation of the mandible eliminates functional stimulus to its growth and that, when the mandible is immobile, the muscles of mastication undergo variable degrees of atrophy of disuse: The

muscles shrink, flatten out, and contribute secondarily to the malformation of the face. It is only by disconnecting the mandible from the fixed bones of the skull—even at the expense of removing the condyle, the coronoid process, and the adjacent segment of the ramus—that the mandible may be permitted to move and catch up with its allotted span of growth under the influence of function. The muscles of mastication, which have laid dormant, will then begin to contract and bulge and thus partly remedy the flattening of the face on the affected side.

CLINICAL MANAGEMENT

Appraisal and Scrutiny

One may question the advisability of mobilizing a solidly fused joint elsewhere in the body, especially in the lower extremity, but in ankylosis of the temporomandibular joint there is no other recourse. Certain points, however, need to be clarified before surgical mobilization of the mandible is undertaken. Preoperatively, the surgeon must ascertain: (1) the condition of the muscles of mastication; (2) whether the ankylosis is unilateral or bilateral and, if bilateral, which side is the seat of primary affection and which has stiffened secondarily; and (3) the type of ankylosis. Other interrelated factors, such as the age of the patient at onset of stiffness, the length of time the jaw has remained ankylosed or interlocked, the mobility of other joints, especially those of the cervical spine, and the nature of the primary disease, must also be taken into consideration, as they modify the management and condition the outcome.

In Marie-Strümpell type of inflammatory arthritis, when both joints have been effaced for a protracted period of time and the muscles of mastication have undergone irreversible changes, surgical mobilization of the mandible accomplishes very little. This is especially true when the cervical spine is involved and the supplementary movement at the atlanto-occipital joint, which adds to the range to which the mouth can be opened by tilting the head backward, is likewise gone. However, even a hopelessly crippled arthritic must open his mouth a bit, and there is no way of telling whether the atrophied muscles are capable of recovery unless they are given a chance by mobilization of the mandible. Often in these patients the thoracic cage is frozen; breathing is mainly abdominal and, to a lesser extent, cervical. The platysma, which serves as an accessory muscle to breathing, is much stronger than in normal individuals. As the platysma originates in the skin of the neck and inserts into the lower border of the mandible, it can, in the absence of stronger depressors of the jaw, pull the mandible down, away from the maxilla, as much as a centimeter. This is a rewarding feat for one who has not opened his mouth for a long period of time.

Of the muscles which protrude and elevate the jaw, only the external pterygoid and the temporalis insert in close relation to the joint and may have been primarily involved in the inflammatory process. The internal pterygoid and the masseter, which are perhaps the two most important muscles of mastication, suffer only atrophy from disuse; by mobilization of the jaw they are given a chance to regain their contractility and to aid in elevating the mandible and in clenching and grinding the teeth, as is needed when one chews solid food. When the mandible is fixed on one side, due to congenital synosteosis, some of the muscles on that side may be fibrotic or completely wanting; but following mobilization of the jaw, the muscles on the opposite side will open and close the mouth. Even though this movement may appear lopsided, it is useful and hence desirable. In unilateral ankylosis of the joint, both the masseter and the internal pterygoid will regain their contractility after use.

In older adults with long-standing ankylosis of the jaw, both sides must often be operated upon before the mandible can be mobilized, even though only one side may have been affected primarily and the other has become stiffened secondarily. On the other hand, when fixation is recent, especially in children and young adults, surgery directed only at the primary seat will suffice. In the absence of leading clues, such as a definite history of

injury to one side of the jaw, localized pain, drainage of the ear, or cellulitis, it is often difficult to determine definitely which of the two joints had been primarily affected and which has become stiffened secondarily. Deviation of the chin, the greater flattening of facial contour, the absence of impulse transmitted to the palpating finger, placed in the external ear as the patient tries to open and close his mouth, and the diminished distance from the lower edge of the zygomatic arch to the inferior border of the ramus may all point to the primary seat. These signs are not altogether reliable, however, nor are they always present. Roentgenograms may reveal narrowing or effacement of the articular space, sclerosis of the bones surrounding the joint, or even shadows indicative of osseous strands, running from the mandible to the temporal bone or maxilla. Arthrograms are worthless.

Ordinarily it matters little whether the fixation across the joint is due to fibrous bands or bony bridges. In pyogenic arthritis, introduced by way of the blood stream or by direct extension of infection from the mouth or middle ear, the ankylosis is usually osseous and firm. There is frequently solid fusion between the mandibular condyle, the base of the skull, and the front of the middle ear. The surgeon who fails to recognize this, and attempts to extract the condyle by forceful twisting and levering, runs the risk of causing a fracture across the base of the skull or through the tympanic portion of the temporal bone into the middle ear. The same precaution must also be taken when ankylosis is due to Marie-Strümpell type of inflammatory arthritis, where the osseous strands across the joint are firmer than the bones they bridge. In both of these instances, it is much safer to forego extracting the condyle and to resect a large segment from the ramus farther down.

Anaesthesia

In the presence of an immobile or interlocked mandible, general anaesthesia can be treacherous. It may not be possible to establish and maintain a clear airway; the mouth cannot be pried open, the tongue pulled, or secretions aspirated from the throat. Local anaesthesia is much safer, and preference should be given to it whenever it is deemed feasible.

In adults, surgical mobilization of the mandible can well be carried out with the aid of local and regional infiltration of novocain. The addition of an appropriate dose of a vasoconstrictor, such as epinephrine, aids hemostasis and counteracts reactions to procaine. The preliminary administration of a sedative, consisting of morphine and atrophine or one of the barbiturates, allays pain and reduces secretions, thereby contributing to the comfort of the patient. In nervous individuals, local anaesthesia should be supplemented with slow infusion of from 0.1 to 0.2 per cent. of sodium pentothal; but sedation should never be forced to the point at which protective reflexes are abolished.

In children and extremely apprehensive adults, there is no alternative to general anaesthesia. The ordinary inhalation mask is a constant source of annoyance to the surgeon when he is working in the vicinity of the face, and fails to assure a patent pathway to the lungs. Occasionally one may have to perform a preliminary tracheotomy and administer the inhalation anaesthetic through the tube within the windpipe. In general, however, nasal intubation is to be preferred. In children, the nasal tube can be inserted following a swiftly induced general anaesthesia. In adults, the nasal catheter is installed after topical application of one of the cocaine derivatives.

After nasal intubation and the establishment of a patent airway, the inhalation agent is administered at some distance from the field of surgery. Narcosis need not be deep and may well be supplemented by local anaesthesia. A tracheotomy set must be available during the induction and emergency phases and for some time after the patient has regained consciousness. The airway may be obstructed as a result of aspiration of secretions, or due to laryngospasm or dropping of the mobilized mandible against the throat. Persons who have not chewed for a long period of time may not have strong enough muscles to

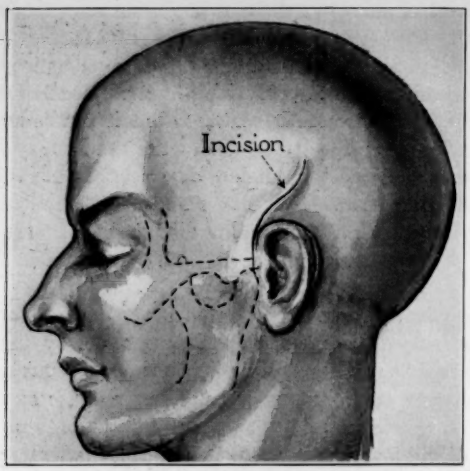

FIG. 1-A

FIG. 1-B

Surgical Mobilization of the Mandible

The articular tubercle of the temporal bone, the adjacent portion of the zygomatic arch, the proximal portion of the ramus of the mandible and its processes are resected through a subperiosteal approach which avoids injury to motor nerves and major blood vessels. As a rule, both temporomandibular joints are operated upon at intervals of two or three weeks.

Fig. 1-A: The skin cut begins about an inch above the proximal pole of the helix; it curves backward, forward, and downward in the form of a question mark. The lower limb of the skin cut descends close to the anterior border of the ear, posterior to the point where the temporal artery can be palpated. It ends at the level of the tragus.

Fig. 1-B: The skin incision is developed down through the temporalis fascia and the muscle to the calvarium.

support the loosened mandible. The jaw must be held up by mild skeletal traction until the muscles of mastication have sufficiently regained their power of contraction. Until that time, a tracheotomy set should be kept in the patient's room.

Incision and Exposure

Practically all the inadequacies of exposure of the temporomandibular joint are due to its proximity to the facial nerve. One must concede that it is not possible to expose this joint widely by a more direct approach without cutting one of the upper branches of the facial nerve. The approach proposed is devious, but it is safe, since it leads to the joint through a route where there are no motor nerves and very few blood vessels.

The *skin incision* begins about two fingerbreadths above the highest point of the helix; it curves down, backward, and then anteriorly, and descends in front of the ear, tracing a question mark. The distal limb of the skin cut is placed slightly posterior to the superficial temporal artery, close to the anterior border of the ear. It ends at the level of the tragus (Fig. 1-A). The incision is developed through the deep fascia (Fig. 1-B). The upper pole of the temporalis fascia is stabbed, and a curved forceps is passed through the slit from above downward. The temporal fascia is split all the way down to the lower end of the incision, between the spreading jaws of the guide forceps. As it approaches the zygomatic arch, the temporal fascia divides into two layers. Between these, surrounded by a pulp of fat and areolar tissue, passes the small middle temporal artery. The more constant and much larger superficial temporal artery lies outside the temporal fascia and muscle. In line with the fascial incision, the temporal muscle is split and its greater anterior portion is lifted off the underlying bone subperiosteally. It is reflected forward. The superficial temporal vessels and the temporal branches of the facial nerve, padded behind by the temporalis muscle, are retracted anteriorly. At the lower end of the incision, the anterior

FIG. 2-A

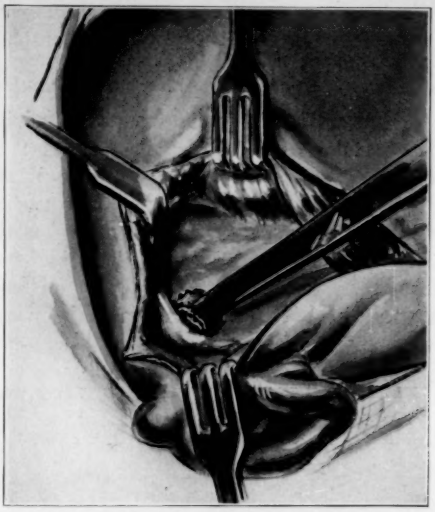

FIG. 2-B

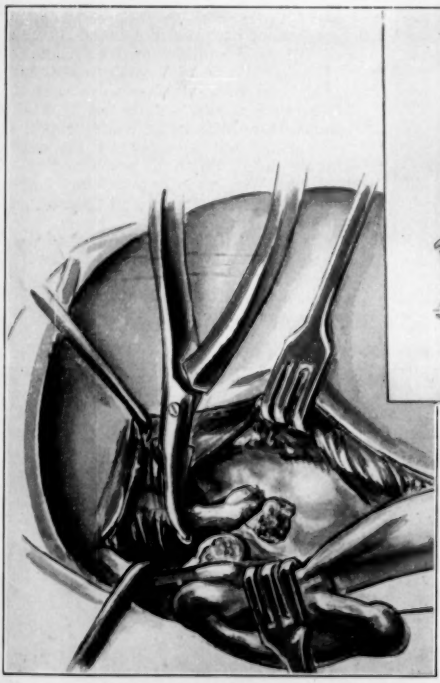

FIG. 2-C

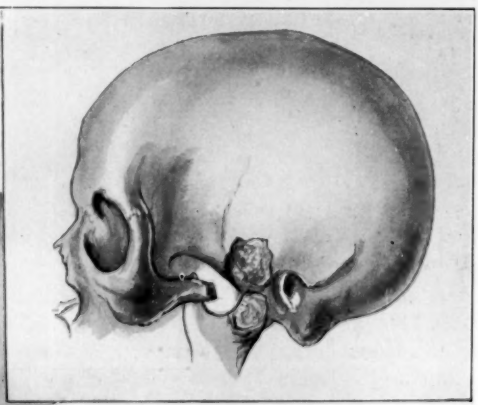

FIG. 2-D

Resection of Articular Tubercle and Posterior Half of the Zygomatic Arch

Fig. 2-A: The articular tubercle of the temporal bone juts out laterally; it constitutes the roof of the temporomandibular joint. The articular condyle of the mandible hides beneath and medial to the articular tubercle. This drawing demonstrates subperiosteal stripping and retraction forward of the temporalis to reveal the zygomatic arch, under which its tendon passes to insert into the coronoid process of the mandible. The dotted line indicates the incision through the capsule. The lateral portion of the capsule of the temporomandibular joint is incised in the line indicated, and the capsule is stripped down over the neck of the articular condyle.

Fig. 2-B: The chisel is placed flush with the squamous portion of the temporal bone and is hammered toward the joint. It disconnects the entire posterior root of the zygomatic arch, and, with it, the articular tubercle of the temporal bone.

Fig. 2-C: The zygomatic arch is bitten off with a rongeur, just in front of the point where it gives origin to the masseter. The anchorage of this muscle is not disturbed.

Fig. 2-D: Shows the extent of the resection of the zygomatic arch and the articular tubercle needed to reveal the fused condyle of the mandible. The long axis of the condyle runs from outside medially and slightly backward.

auricular artery leaves the superficial temporal vessel and passes backward. If more room is needed downward, it is best to isolate this small twig and ligate it.

Resection of Articular Tubercle and Adjacent Zygomatic Arch

Exposure of the temporomandibular joint is sought through the musculoskeletal and trans-skeletal plane. The temporal muscle is traced down to its insertion into the coronoid process; the diverging limbs of the temporalis fascia, which connect with the inner and outer borders, respectively, of the zygomatic arch, are stripped forward (Fig. 2-A). The articular tubercle (eminentia articularis) of the temporal bone, which constitutes the posterior root of the zygomatic arch, is laid bare along its superior aspect. Inferiorly, the articular tubercle and the adjacent portion of the zygomatic arch are cleared subperiosteally off the temporomandibular ligament and the joint capsule. When the mouth is closed, as is the case in temporomandibular ankylosis, the condyle hides behind and medial to the articular tubercle. A good view of the temporomandibular joint cannot be obtained unless the overlying bony prominence is chiseled off. A sharp osteotome is placed against

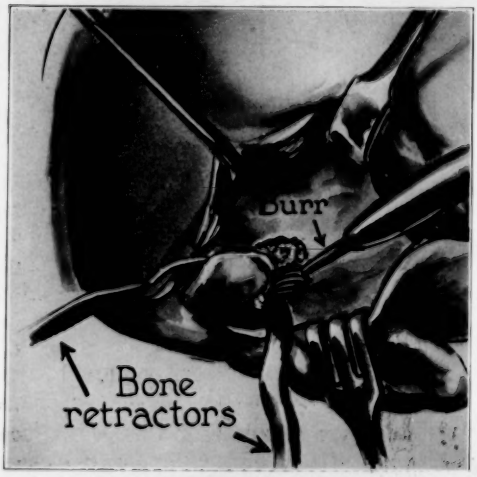

FIG. 3-A

Resection of Proximal Portion of the Ramus of the Mandible and Its Process

Fig. 3-A: Two small curved periosteal retractors are passed around the neck of the condyle, one in front and one behind, care being taken not to penetrate the external auditory meatus with the posterior periosteal retractor. With a motor burr, the condyle of the mandible is softened and then bitten off with a rongeur.

Fig. 3-B: The coronoid process of the adjacent proximal portion of the ramus is also resected, either with a rongeur or with a chisel.

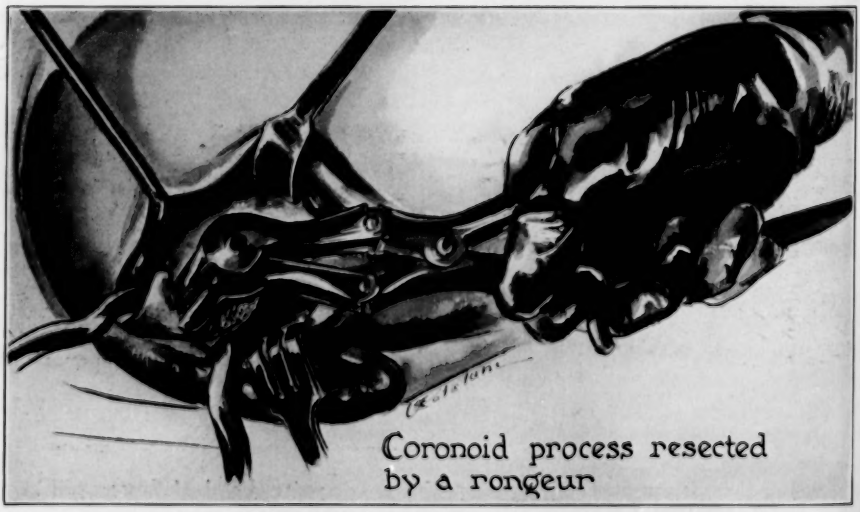

FIG. 3-B

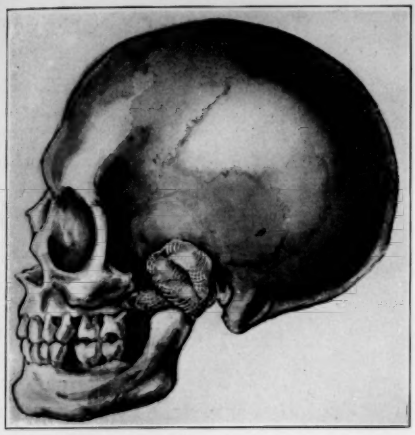

FIG. 3-C

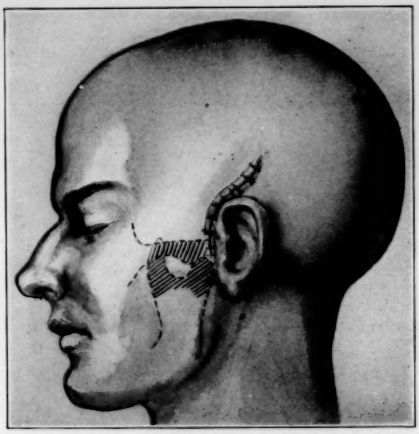

FIG. 3-D

Fig. 3-C: A space which will admit the surgeon's thumb is created; this space is packed with oxycel gauze.

Fig. 3-D: Shows the wound closure. The shaded area indicates the extent of bone resection.

the posterior root of the zygomatic arch, flush with the outer surface of the squamous portion of the temporal bone. The lateral roof of the temporomandibular socket, including the articular tubercle, is shaved off. The zygomatic process, posterior to the origin of the masseter, is severed (Fig. 2-B). When the resected tubercle and the adjacent portion of the zygomatic arch have been removed, the articular condyle can be seen and the nature of its fusion with the temporal bone can be ascertained (Figs. 2-C and 2-D).

Resection of the Proximal Portion of the Ramus of the Mandible and Its Processes

In general, the more bone resected from the proximal portion of the ramus of the mandible and its processes, the better. Paradoxical as it may seem, a greater amount of bone must be resected in children, where osteogenesis is more exuberant. In ankylosis due to pyogenic joint disease and Marie-Strümpell type of inflammatory arthritis, resection of a segment across the waist of the ramus is a safer procedure than dislodging the solidly fused condyle by force. Ordinarily, both the coronoid and condylar processes and the adjacent portion of the proximal part of the ramus are excised. When the coronoid process is resected, the temporalis, which inserts into it, loses its functional usefulness. (The func-

FIG. 4-A

FIG. 4-B

Skeletal Traction

Both as a preventive measure after injuries and postoperatively, following surgical mobilization of the jaw, traction through the mandible itself is regarded as expedient.

Fig. 4-A: Traction through the body of the mandible with the aid of a Crutchfield tong.

Fig. 4-B: Traction through the chin by a Kirschner wire and countertraction by a Crutchfield tong hooked to the skull.

Skeletal Traction in Preventing Stiffness at the Temporomandibular Joint

In fracture-dislocations and crush fractures of the mandibular condyle and in depressed comminuted fractures of the zygomatic arch, timely use of skeletal traction may obviate otherwise inevitable immobility of the jaw.

FIG. 5-A

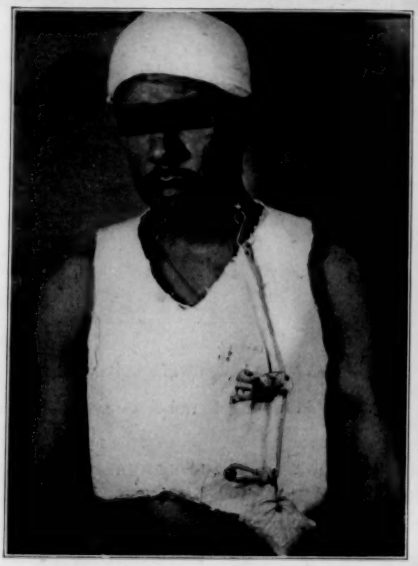

FIG. 5-B

Unilateral Traction by Mattress Wire in Fracture-Dislocations of the Temporomandibular Joint

Fig. 5-A: Lateral roentgenogram of the mandible shows a twisted mattress wire which passes through the angle of the mandible and connects to a traction hook outside.

Fig. 5-B: Showing the Minerva jacket and the weight-and-pulley arrangement over the breastpiece. The plaster cast prevents the head from tilting forward and avoids strain on the cervical spine.

FIG. 5-C

FIG. 5-D

Fig. 5-C: Profile view with the mouth closed against the resistive force of a three-pound weight and gravity.

Fig. 5-D: Indicates the opening of the mouth.

tion of the temporalis in elevating the jaw depends mainly upon the articular condyle, which serves as the fulcrum to a second-class lever.)

FIG. 6-A

FIG. 6-B

Unilateral Traction by a Paddled Screw Inserted Vertically into the Angle of the Mandible
This man had a comminuted fracture of the body of the mandible and fracture-dislocation of the right condyle.

Figs. 6-A and 6-B: Frontal and profile views of the Minerva jacket and arrangement for screw-and-pulley traction. The paddle of the screw is connected to the head piece of the plaster jacket and traction is applied mid-way on the screw, converting it to a second-class lever.

FIG. 6-C

FIG. 6-D

Opening of the mouth, two months after injury.

Preliminary to the excision of the proximal portion of the ramus and its processes, the capsular ligaments and the periosteum are stripped downward, so that the bone can be resected subperiosteally. The parotid gland sends a tongue upward, which curls around and fills the space behind the condyle and in front of the external acoustic meatus. The facial nerve enters the main body of the parotid gland farther down. In stripping the superior pole of the parotid and the periosteum off the posterior border of the mandible, care should be taken not to penetrate the external acoustic meatus. The anterior wall of

FIG. 7-A

FIG. 7-B

FIG. 7-C

FIG. 7-D

Postoperative Use of Skeletal Traction in Children

Skeletal traction by a Kirschner wire through the chin is regarded as an important adjunct to the surgical mobilization of the temporomandibular joint. Immediately after surgery, it holds the chin forward and prevents the loosened jaw from dropping backward and choking the patient; in the ensuing three to four weeks it stretches the contracted soft tissues and maintains, if not augments, the distance between the raw surfaces caused by resection of bone; it creates tension, against which the atrophic muscles of mastication can be made to contract and to regain their strength. In children, one runs the risk of causing dislocation of the cervical spine; it is best to immobilize the neck in a Minerva jacket. The breastpiece of the plaster jacket anchors the base of an adjustable pulley.

Figs. 7-A and 7-B: Views of the Minerva jacket and the traction-pulley arrangement.

Figs. 7-C and 7-D: Close-up views, showing the opening of the mouth.

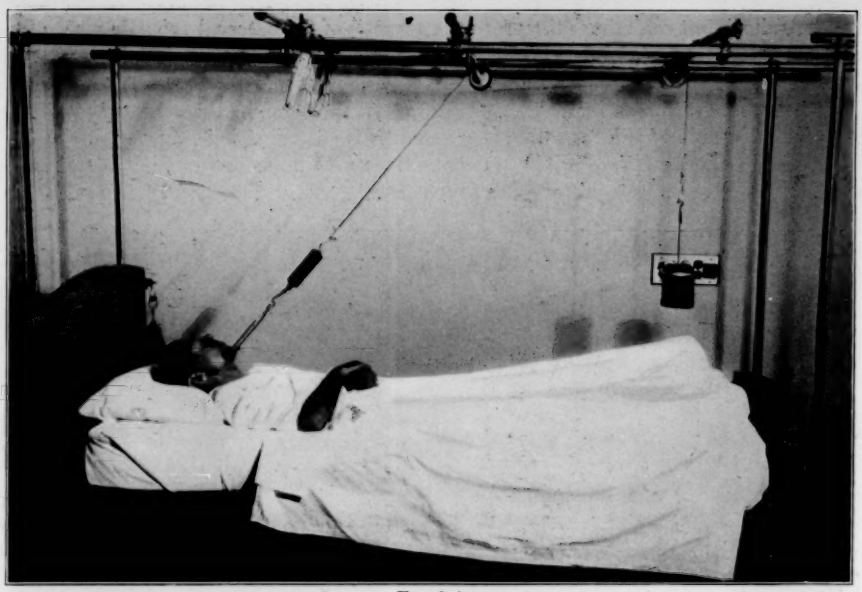

FIG. 8-A

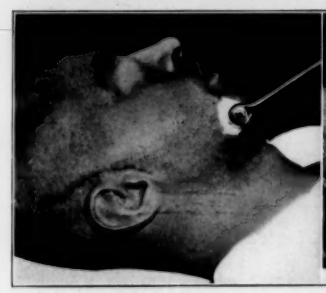

FIG. 8-B

FIG. 8-C

FIG. 8-D

Postoperative Skeletal Traction in Adults

Where there are good neck muscles and no active arthritis is present in the cervical spine, as much as thirteen pounds of weight may be tolerated through the chin when the patient is recumbent, and three to four pounds when he is up.

Fig. 8-A: Photograph of the patient in bed with traction through the chin. A spring scale, serving as a link in the line of traction, registers the actual pull in pounds.

Fig. 8-B: Close-up profile view showing the Kirschner wire through the chin.

Figs. 8-C and 8-D: Show the patient actively contracting his muscles of mastication against tensile stress, and with the mouth open.

Fig. 8-E: Frontal view shows the traction when the patient is up. A three-pound weight is attached to the horseshoe which connects with the Kirschner wire in the chin.

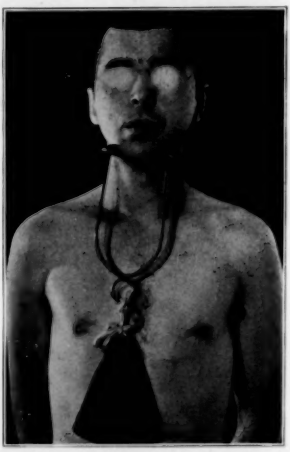

FIG. 8-E

the auditory canal can also be torn by vigorous pulling of the ear backward. In either instance, it is best not to suture the rent. At the termination of the operation, the canal is packed with a wick of oxycel gauze, down to the tympanic membrane. Ordinarily

these tears heal, but sometimes they result in stenosis of the external acoustic meatus and impairment of hearing. With the subperiosteal approach described, the facial nerve, which passes through the substance of the parotid gland, is not injured.

It is a matter of individual preference as to how the condyle, the coronoid process, or the proximal ramus of the mandible is severed. With the incision advocated, the Gigli saw is hard to handle. It is possible to cut the bone with an osteotome, but better still is the combination of a burr and rongeur. An electric burr with an acorn tip is used to bore

FIG. 9-A

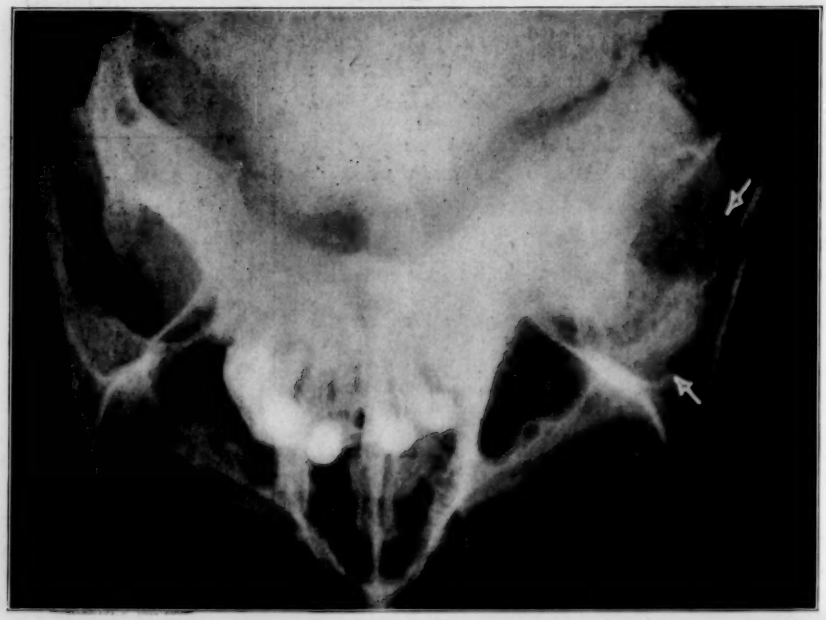

FIG. 9-B

FIG. 9-C

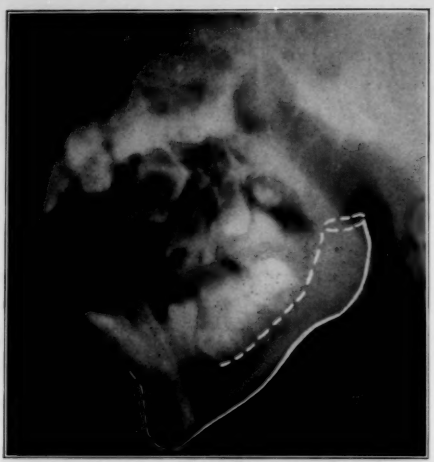

FIG. 9-D

Case 1. Unilateral congenital synosteosis in a girl, aged ten. At birth the patient's mouth could not be opened. When the lower jaw was forced down, the mandible was fractured on the left side. At the age of four, she was given ether anaesthesia and an unsuccessful attempt was made to pry the jaws apart. The child was fed through a hole left by a missing tooth. When she was six and one-half years old, an intra-oral operation was performed and, according to the report, the bridge of bone between the mandible and maxilla was resected. The day after the operation the mouth opened one inch, but began to close again within twenty-four hours. It had closed tightly in one month. The child was operated upon by the author on April 29, 1948, when she was nine and one-half years old. At operation, the coronoid process of the left mandible was found to be fused to the left zygomatic arch; the ramus was continuous with the maxilla in front, and there was a tight band extending from the styloid process of the temporal bone to the angle of the mandible; the left temporal muscle was completely absent.

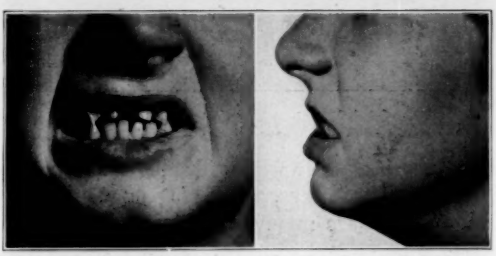

FIG. 9-E FIG. 9-F

FIG. 9-G FIG. 9-H

Fig. 9-A: Preoperative lateral roentgenogram of the left temporomandibular joint shows complete osteosynthesis between the ramus of the mandible and the maxilla in front, and between the coronoid process and the zygomatic arch. The arrows point to a solid mass of bone.

Fig. 9-B: In occipitomental view the solid mass of bone is seen on the left side.

Figs. 9-C and 9-D: Postoperative lateral roentgenograms with the mouth closed and open.

Figs. 9-E and 9-F: Preoperative photographs show the great muscular effort required to open the mouth.

Figs. 9-G and 9-H: Four months after surgery. Three months later the child's mouth opened one-quarter of an inch more; there was good side-to-side grinding or pterygoid action.

several holes through the outer plate of the condyle or the ramus, and the septa between these are bitten off with a rongeur (Fig. 3-A). After the inner table has been cut, a curved periosteal elevator is passed from above downward, in order to protect the internal maxillary artery; this passes medial to the bone, but is separated from it by the collateral ligaments and, farther forward, by the external pterygoid. The jaw is pushed up over the periosteal elevator, and the proximal portion of the ramus of the mandible and its processes are nibbled until a space not less than two centimeters is created (Fig. 3-B).

FIG. 10-A FIG. 10-B

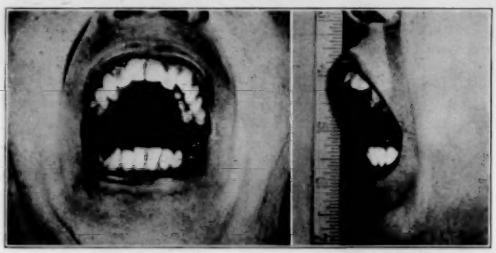

FIG. 10-C FIG. 10-D

Case 2. Bilateral interlocking due to old arthritis, possibly of traumatic origin. The patient was a man, twenty-eight years of age. At the age of ten he had been hit by an automobile and his "jaw" had been broken. It was set, but he could not open his mouth afterward. The lower jaw receded and he fed himself through the space left by malocclusion. A roentgenographic report on December 6, 1947, was as follows: "Examination of the temporomandibular joints in open and closed positions reveals relatively little motion". The right temporomandibular joint was operated upon December 5, 1947; the left, January 5, 1948.

Figs. 10-A and 10-B: Preoperative photographs show the extent of opening of the mouth and the space left by malocclusion.

Figs. 10-C and 10-D: Photographs six months after operation on the left side, and seven months after operation on the right.

Where the condyle has not firmly fused to the temporal bone, it is removed; where there is bony fusion between the posterior border of the ramus and the petrous or tympanic portion of the temporal bone, more bone is excised from the ramus; in congenital synosteosis between the mandible and maxilla, more bone is resected from the anterior border of the ramus. In all instances, a finger is passed into the space created by the resection of bone. All bone chips are removed and sharp spicules are smoothed. The articular socket of the temporal bone sends an apron-like projection medially, which descends almost two centimeters downward. It is best to resect this portion of the temporal bone, but to leave the roof of the socket undisturbed. The cavity created is packed with oxycel gauze snugly, but not tightly (Fig. 3-C). This pack prevents any possible contact of the raw surfaces of the bones; it fills the space between them and obviates oozing and accumulation of blood and exudates. Oxycel gauze is said to retard osseous proliferation. The temporal fascia and the skin are closed over the oxycel pack (Fig. 3-D).

After-Care

When general anaesthesia is given, the patient must be protected from choking himself immediately after the operation. This accident sometimes occurs after surgery of the lower jaw, and can well be obviated by skeletal traction, which prevents the mandible from dropping back against the throat. When one temporomandibular articulation is free and the other is fused, skeletal traction is instituted immediately after the surgical mobilization of the ankylosed joint. When both sides are fused, one joint is mobilized at a time with a period of two to three weeks intervening. During this interval the ankylosed articulation acts as a splint for the joint which has been operated upon, and skeletal traction is not needed. However, traction should be instituted at the termination of the second operation, and the patient should not be allowed to go through a night without traction until he has regained muscular control of his jaw.

Skeletal Traction

This serves several other useful purposes: It keeps the raw surfaces of bone distracted, and thereby prevents reunion; it pulls the chin forward and holds it there until the cut end of the ramus becomes sealed over in its new surrounding; and, finally, it offers the atrophic muscles of mastication a tensile stress against which they may contract and regain

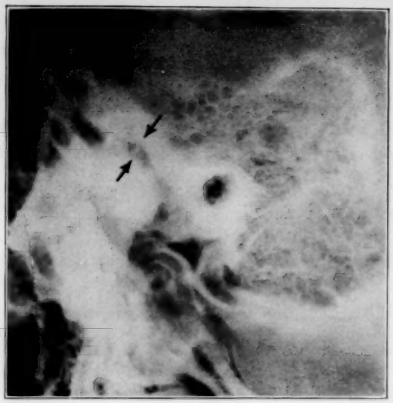

FIG. 11-A

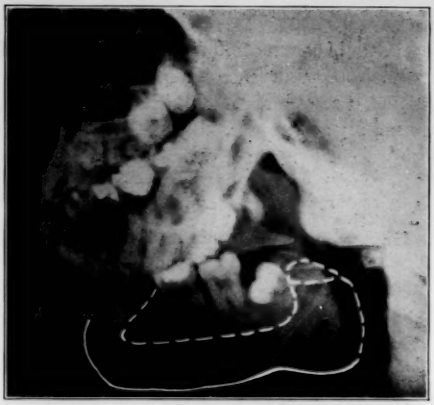

FIG. 11-B

Case 3. Unilateral bony ankylosis due to pyogenic arthritis. The patient was a woman, aged eighteen. At the age of six, she had had a sore throat which was followed by septic arthritis of both hips, both knees, and the right temporomandibular joint. Drainage from these joints occurred intermittently until about the age of twelve.

Fig. 11-A: Lateral roentgenogram of the right temporomandibular joint. Note the extensive sclerosis and almost complete effacement of the articular space between the arrows.

Fig. 11-B: Lateral roentgenogram showing the resection of the mandibular ramus. Note that more bone has been taken from the ramus farther down.

In pyogenic joint disease and in the Marie-Strümpell variety of inflammatory arthritis, where the ankylosis at the joint is firm, it is best to resect more bone from the mid-portion of the ramus than proximally.

Figs. 11-C and 11-D: Preoperative photographs show the extent of opening of the mouth.

Figs. 11-E and 11-F: Postoperative photographs, taken eight months later, show the degree to which the mouth can be opened actively.

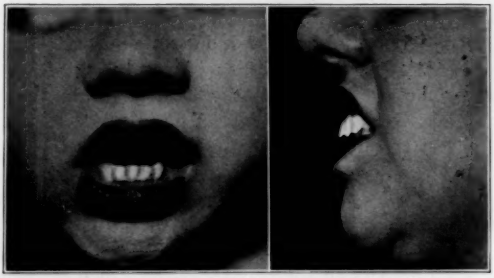

FIG. 11-C FIG. 11-D

FIG. 11-E FIG. 11-F

their power (Figs. 4-A to 8-E).

In fracture-dislocations of the mandibular condyle, with interlocking at the joint, unilateral traction may be applied through the angle of the jaw by either wire or screw (Figs. 5-A to 6-D). After surgical mobilization of the jaw, traction through the chin is better, since it overcomes recession and prevents the mandible from dropping back against the throat. Occasionally in Marie-Strümpell arthritis, some of the segments of the cervical spine may be the seat of inflammatory activity and yet remain unfused; skeletal traction through the mandible will cause strain and result in added pain in the cervical spine, unless counteraction is provided by traction upward from the skull or by a head-and-torso plaster jacket.

In children the cervical vertebrae become dislocated with greater ease. Here skeletal traction through the mandible may cause subluxation at the atlanto-axial segment; as a

precautionary measure, the head and the torso are encased in a plaster jacket and the forehead is prevented from tilting forward (Figs. 7-A to 7-D). Adults with a normal cervical spine and strong muscles will tolerate as much as thirteen pounds of traction through the chin. For children, three to four pounds will suffice. Traction is maintained for as long as the wire through the mandible is tolerated,—usually for about five weeks. The weight is reduced when the patient is up and gravity comes into play (Figs. 8-A to 8-E).

SUMMARY

A method has been propounded for mobilization of an ankylosed or interlocked temporomandibular joint. It consists of two basic tenets: (1) resection of the articular ends of bones and (2) skeletal traction. For surgical mobilization of the mandible, it is important that the incision should avoid the larger blood vessels and the upper branches of the facial nerve; that the temporomandibular joint be approached from above and back, downward and forward subperiosteally, under the temporalis and through the base of the articular tubercle of the temporal bone; that a wide segment of bone be resected from around the joint, mostly from the mandible, so as to create a space; and that the cavity excavated be packed by some material which will prevent seepage and hematoma and keep the raw surfaces of bone from coming together and reuniting. Oxycel gauze is both hemostatic and slowly absorbable, and has been shown to counteract proliferation of bone; it is well suited for use as a pack. If possible, the cavity created should not communicate with the ear. If the external acoustic meatus has been perforated inadvertently, it is best not to suture the rent, but rather to pack the ear canal up to the tympanic membrane with a wick of oxycel gauze.

The pattern of bone resection in and around the temporomandibular joint admits of several variations:

1. When the articular condyle has become firmly fused to the temporal bone, resection of a segment from the ramus farther down is deemed safer than gouging the condyle out of its solid matrix at the base of the skull.

2. In pyogenic arthritis, caused by extension of infection from the middle ear, more bone is chiseled off the posterior border of the ramus in order to gain a wider space between the mandible in front and the tympanic plate behind.

3. In congenital synosteosis between the mandible and the maxilla, more bone is excised from the anterior border of the ramus.

Skeletal traction through the mandible is considered beneficial for injuries to the temporomandibular joint and after operation. It is suggested that, in some fractures and dislocations of the articular condyles and in comminuted depressed fractures of the zygomatic arch, the timely institution of skeletal traction will prevent the fixation of the jaw which often follows these injuries. After surgical mobilization of the mandible, skeletal traction through the chin is regarded as expedient; it has many advantages over the old-fashioned wedge or mouth gag, which exerts only static pressure against the teeth. The stress caused by skeletal traction is dynamic, and the pin through the mandible is well tolerated. Skeletal traction augments the space created by excision of bone; it keeps the raw surfaces of bone apart, and aids the muscles of mastication to contract against tension and regain their strength. Moreover, when both temporomandibular joints are subjected to resection and the muscles of mastication are weak, as they invariably are in long-standing immobility of the mandible, the jaw drops toward the throat and makes breathing difficult. The loosened mandible must be lifted away from the throat and held up by skeletal traction.

REFERENCES

1. Abbe, Robert: An Operation for the Relief of Anchylosis of the Temporo-Maxillary Joint, by Exsection of the Neck of the Condyle of the Lower Jaw; with Remarks. New York Med. J., **31**: 362–366, 1880.

2. Bottini, Enrico: Asportazione sottoperiostea a'ambo i condili della mandibola per serramento stabile della bocca. Gior. d.r. Accad. di Med. di Torino, **35**: 549–558, 1872.
3. Bottini, Enr.: Resektion beider Kondylen des Unterkiefers wegen Mundsperre. Wiener Med. Wchnschr., **22**: 695, 1872.
4. Brash, J. C.: A Preliminary Note on the Mode of Growth of the Mandible. British Dent. J., **50**: 611–616, 776, 1929.
5. Brodie, A. G.: On the Growth Pattern of the Human Head from the Third Month to the Eighth Year of Life. Am. J. Anat., **68**: 209–262, 1941.
6. Dieffenbach, J. F.: Die operative Chirurgie, Band 1. Leipzig, F. A. Brockhaus, 1848.
7. Eggers, G. W. N.: Arthroplasty of the Temporomandibular Joint in Children with Interposition of Tantalum Foil. A Preliminary Report. J. Bone and Joint Surg., **28**: 603–606, July 1946.
8. Esmarch, Friedrich: Beiträge zur praktischen Chirurgie. 2 Heft: Die Behandlung der narbigen Kieferklemme durch Bildung eines kündstlichen Gelenkes im Unterkiefer. Kiel, Schwers, 1860.
9. Esmarch, Frédéric: Traitement du resserrement cicatriciel des mâchoires par la formation d'une fausse articulation dans la continuité de l'os maxillaire inférieur. Arch. Gén. de Méd., **15** (5 série): 44–56, 1860.
10. Goodwillie, D. H.: Ankylosis of Temporo-Maxillary Articulation of Long Standing; Fracture of Right Condyle; Atrophy of the Depressors and Contraction of the Elevator Muscle of the Inferior Maxillary. Monthly Rev. Dent. Surg., **4**: 226–231, 1875.
11. Gross, S. D.: A Case of Fibrous Tumour of the Lower Jaw, in which the Left Half of That Bone was Successfully Removed at the Temporo-Maxillary Articulation. Am. J. Med. Sciences, **16**: 344–346, 1848.
12. Gross, S. D.: A System of Surgery. Pathological, Diagnostic, Therapeutic, and Operative. Philadelphia, Blanchard and Lea, 1859.
13. Heath, Christopher: Injuries and Diseases of the Jaws: The Jacksonian Prize Essay of the Royal College of Surgeons of England, 1867, Ed. 3, p. 406. London, Churchill, 1884.
14. Helferich: Ein neues Operationsverfahren zur Heilung der knöchernen Kiefergelenksankylose. Arch. f. Klin. Chir., **48**: 864–870, 1894.
15. Humphry, G. M.: Excision of the Condyle of the Lower Jaw. Assn. Med. J., **4**: 61–62, 1856.
16. Lexer, E.: Ueber hautimplantation an stelle der freien faszienplastik. Jahresb. d. Ärztl. Ver. zu Frankfurt a.M., S. 64–67, 1913.
17. Pauly (Clinique de Lisfranc): Sur le cancer de la partie inférieure de la face. Résection de l'os maxillaire inférieur. Bull. Clin., Paris, **1**: 314–319, 463–468, 1835–1836; **2**: 13–18, 73–80, 201–210, 1836–1837.
18. Little, J. L.: Excision of Lower Jaw for Osteo-Sarcoma; Recovery. New York Med. J., **18**: 614–619, 1873.
19. MacAusland, W. R., and MacAusland, A. R.: The Mobilization of Ankylosed Joints by Arthroplasty. Philadelphia, Lea and Febiger, 1929.
20. Mazzoni, C., e Tamburini, A.: Lussazione bilaterale del mascellare inferiore antica e irreducible; resezione dei due condili; successo completo. Gazz. Med. di Roma, **3**: 37–39, 1877.
21. Mikulicz, J., und Kümmel, W.: Die Krankheiten des Mundes. Jena, Gustav Fischer, 1898.
22. Mott, Valentine: Case of Osteo Sarcoma in Which the Right Side of the Lower Jaw Was Removed Successfully after Tying the Carotid Artery. New York Med. and Phys. J., **1**: 385–394, 1822.
23. Murphy, J. B.: Arthroplasty for Intra-Articular Bony and Fibrous Ankylosis of Temporomandibular Articulation. Report of Nine Cases. J. Am. Med. Assn., **62**: 1783–1794, 1914.
24. Pickerill, H. P.: Ankylosis of the Jaw: Cartilage Graft Restoration of the Joint: A New Operation. Australian and New Zealand J. Surg., **11**: 197–206, 1942.
25. Rizzoli, Francesco: Risecazione della mascella inferiore. Bull. delle Scienze Med., **8**: 80–85, 1839.
26. Rizzoli, Francesco: Storia di una immobilità della mascella inferiore trattata con un nuovo processo operatorio. Bull. delle Scienze Med., **9** (Série 4): 81–89, 1858.
27. Rizzoli, Francesco: Di un'ablazione completa intrabuccale e sottoperiostea della mascella inferiore. Bull. delle Scienze Med., **19**: 42–46, 1863.
28. Syme, James: Case of Osteo-Sarcoma of the Lower Jaw. Edinburgh Med. and Surg. J., **30**: 286–290, 1828.
29. Syme, James: Disarticulation of the Ramus of the Lower Jaw, without Opening the Cavity of the Mouth. London and Edinburgh Monthly J. Med. Science, **3**: 963–965, 1843.
30. Verneuil, A.: De la création d'une fausse articulation par section ou résection partielle de l'os maxillaire inférieur, comme moyen de remédier à l'ankylose vraie ou fausse de la machiore inférieure. Arch. Gén. de Méd., **15** (5 série): 174–195, 284–316, 1860.

TRIALS AND TRIBULATIONS IN ATTEMPTED FEMORAL LENGTHENING *

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The development of surgical procedures for the correction of inequality of limb length closely parallels the development of orthopaedic surgery itself. Advance in operative surgery is, as a rule, synonymous with simplification, and equalization of limb length is no exception. The more difficult procedures of femoral shortening and femoral lengthening were advocated first. Abbott, in 1927, reported his operation for lengthening of the tibia and fibula, and Phemister, in 1933, reported his operation for epiphyseal arrest. By comparison, Phemister presented a very simple operation, and during the past fifteen years its almost universal use has reduced to a small proportion the number of major operations performed for equalization. Still more recently, the experimental work of Haas has made possible temporary closure of the epiphyses, and the recent report of Blount and Clarke indicates that accurate growth control can be assured by this relatively simple surgical procedure.

In spite of these rapid strides in the simplification of measures designed to reestablish limb length, we still encounter an occasional individual who is too old for surgery on the epiphyses and in whom femoral lengthening seems to be the most desirable procedure from a clinical standpoint. An individual (particularly a boy) who is extremely short can hardly sacrifice more height through femoral shortening without feeling that he has been handicapped needlessly. If the shortening is largely or entirely in the femur, lengthening of the tibia results in a peculiar appearance, because the knees are at different levels. In the occasional case, therefore, lengthening of the femur still represents the most desirable procedure.

After the operation for femoral lengthening has been chosen, the big problem has always been how best to attempt the actual bone lengthening. Many technical variations have been used, and several of them will be discussed here.

Codivilla, in 1905, reported an attempt at femoral lengthening by an oblique femoral osteotomy and skeletal traction through the calcaneus. Traction was applied briefly with the patient under anaesthesia, and the increased length was maintained by immediate application of a plaster spica. Codivilla attempted to repeat the application of traction at regular intervals. He reported twenty-six cases treated in this manner, eleven of which were malunited fractures. The gain in length varied between 3 and 8 centimeters. Freiberg, in 1912, reported a case treated by this method. Magnuson [12] reported fourteen cases of operative lengthening of the femur in which traction was applied for twenty to thirty minutes by means of a Hawley table. Magnuson reported considerable shock from the operation, and one of his patients died.

Putti made a definite contribution in 1921, when he reported the use of traction and countertraction, applied directly to the femur. He used one pin above the site of osteotomy and one below, inserting them not only through the lateral aspect of the thigh, but through both cortices of the femur; the two pins were connected with a telescopic tube and a spring extension mechanism. Abbott and Crego, in 1928, experienced considerable difficulty with Putti's method, stating that the pins usually pulled out of the cortex and that control of the position of the fragments was difficult or impossible. Shortly thereafter, Abbott placed

* Read at the Annual Meeting of The American Orthopaedic Association, Colorado Springs, Colorado, May 19, 1949.

the operation on a more sound technical and scientific basis by the development of the following methods:

1. Insertion of the pins through the entire femur and thigh to prevent them from pulling out.

2. Insertion of the upper pin in a vertical rather than a horizontal plane to avoid possible injury of the major vessels. (Abbott credits E. C. Bull, M.D., with this idea.)

3. The use of three pins, one above and two below, in an attempt to maintain better control of the fragments.

4. The use of four pins, two above and two below, for the same reason.

5. The use of a single-unit extension mechanism by means of coil springs on parallel, threaded bars.

6. The use of the Hoke traction plaster splint with skeletal traction through a single pin.

Even with these advances in technique, difficulties were encountered. From this point we have proceeded by a trial-and-error method, in an attempt to discover some means by which the operation could be more safely and accurately performed. The story of these trials and errors, and of the difficulties and catastrophes which followed, is presented in this paper.

More recent reports in the literature include those of Compere and of Bost. Compere, in 1936, advised the simultaneous insertion of a cortical graft to bridge the defect resulting from bone lengthening, in an attempt to decrease the incidence of non-union. True non-union, however, has not been encountered in the series reported here. Bost, in 1944, described a four-pin method for femoral lengthening, a spring extension mechanism being used on parallel threaded bars. He described his own operative technique which, in addition to a Z-shaped osteotomy, included extensive division of the soft tissues in order to decrease the resistance of these structures. Other than this, his method differed from that of Abbott primarily in the direction of the two pins used in the proximal fragment. Bost used one pin vertically and one obliquely.

The ability to gain increased bone length in this series of cases has not been a major problem. Instead, the principal difficulty has been inability to maintain adequate alignment and opposition of the two fragments.

Abbott performed the first operation for femoral lengthening at the St. Louis Unit of the Shriners' Hospital for Crippled Children on January 18, 1926. In that instance the upper pin was inserted only through the anterior surface of the thigh and it immediately pulled out (Fig. 1-A). The lengthening was continued by straight skeletal traction through the distal pin, and an increase of two inches in length was obtained (Figs. 1-B and 1-C). In the second case, for which the operation was performed on July 19, 1926, two pins were also used, but both were inserted transversely through the entire diameter of the femur and thigh. The upper pin was inserted from within outward, through an incision which descended inferiorly from the origin of the adductor longus for a distance of three to four inches. The femoral vessels were lifted anteriorly and the pin was drilled through the adductor muscles, the shaft of the femur, and the tissues on the lateral aspect of the thigh. This incision was then closed and the remainder of the operation was carried out in the usual manner. In his sixth case, for which the operation was performed on November 29, 1926, four pins were used, two above and two below the osteotomy site. The upper pins were inserted transversely, as already described. Because control of the fragments was still difficult with the use of four pins, Abbott continued to experiment with two pins and occasionally with four.

In his seventeenth femoral lengthening, performed on April 15, 1929, Abbott used only one pin with skeletal traction in a Thomas splint after an oblique osteotomy. Three inches in length were obtained, but complete sciatic paralysis followed, and recovery from this required several months. In his twenty-first case, for which operation was performed

FIG. 1-A

FIG. 1-B

Fig. 1-A: Abbott's first case of femoral lengthening by the method described by Putti. The upper pin, inserted through only one cortex, readily pulled out. Straight skeletal traction was continued through the lower pin and a gain of two inches was obtained.

Fig. 1-B: A fracture subsequently occurred, but healed readily. This shows the degree of union present at twenty-seven months.

Fig. 1-C: Preoperative and postoperative photographs of patient. Two inches in length were gained.

FIG. 1-C

on April 21, 1930, one pin was used below the Z-shaped osteotomy and traction was obtained by the Hoke method, the well leg being utilized for countertraction in a plaster spica. An increase in length of two and one-half inches was obtained.

The next operation was attempted in a similar manner, but failure resulted. This operation was performed by Bull, who was visiting at the time; heavy fish-line silk was tied about the bone fragments at the osteotomy site in an attempt to maintain alignment. No separation of the fragments was obtained and callus formed readily. One month later Abbott reopened the incision at the osteotomy site, but still only one inch of added length was obtained. The same procedure was tried in another case, but pressure sores developed while the limb was in the cast, and only one inch of increased length resulted. Still another case was treated in a similar way, flexible wire loops being used to maintain

alignment of the fragments, but difficulty was encountered in getting the fragments to separate.

A total of thirty-seven cases have been treated by surgical lengthening of the femur at this Hospital; twenty-six of these operations were performed by Abbott, including the one in which Bull also participated. The last operation which Abbott did was performed on November 24, 1930, so that the twenty-six cases were treated over a period of approximately five years. The maximum gain in length was three and one-half inches; the minimum was one inch. An average increase in length of 2.1 inches was obtained. Only eleven cases have been treated by femoral lengthening in the eighteen-year period since that time. Five of these operations were performed by C. H. Crego, while the author has performed the last six.

In Abbott's series of twenty-six cases, several serious complications were encountered. Most of these have been mentioned in his earlier reports and have been adequately discussed by Compere. Pronounced angulation of the fragments occurred in eight cases, infection of the pin holes in five, and sciatic paralysis with ultimate recovery in five. Refracture at the same site, either immediately or within six months after operation, occurred in four cases; marked stiffness of the knee followed in two cases; and there was one case each of operative-wound infection with osteomyelitis, fat embolism, flare-up of an old osteomyelitis, and acute postoperative endocarditis. In one case the upper pin pulled out, and in another the hip on the same side was dislocated by traction.

These problems prompted Crego to use the Hoke well-leg countertraction principle in three of his five cases. Even then it was almost impossible to keep the fragments in line, and the final increase in length was always obtained by simple skeletal traction in a Thomas splint. Since in each case simple skeletal traction was ultimately used, it seemed logical to use such traction primarily. This method had been reported in 1933 by Haas, who used a high oblique osteotomy. He recommended the lengthening with simultaneous correction of a coexisting coxa vara. We observed one case which had been treated by Haas and he, too, had encountered great difficulty in maintaining alignment of the fragments. Nevertheless, skeletal traction was next tried in our series, in conjunction with a supracondylar Z-type of osteotomy. Complete separation of the fragments followed, and great difficulty was encountered in preventing them from actually producing a compound wound. An increase in length of two inches was obtained, however. It was obvious that some more accurate means of controlling the fragments was essential before this operation could be undertaken with safety and with assurance of success.

The next step in this trial-and-error process was the attempted use of a method which, although unknown to us, had previously been described by Magnuson. Had we been aware of his work at that time, we would undoubtedly have avoided the catastrophe which followed. With traction exerted while the patient was on the operating table, at the time of a Z-shaped supracondylar osteotomy, a gain in length of about one and one-half inches was obtained over a period of twenty to thirty minutes. The fragments were then held in position by three screws (Fig. 2-A). No immediate complications were encountered and the circulation seemed adequate at the time the plaster cast was applied. Sensation in the toes was absent following the operation, and twenty-four hours later there was evidence of impaired circulation in the foot and toes; this necessitated splitting of the plaster cast. At the end of forty-eight hours the entire leg was cold below the knee, arterial pulsations had disappeared from the foot, and a distinct color change was apparent just below the osteotomy site.

The patient was returned to the operating room at this time; the wound was opened; the screws were removed; and the fragments were replaced in their original position and again fixed with screws. There was no improvement in the circulation, however, and at the end of seventy-two hours there was evidence of gas in the tissues about the foot and ankle, although no gas had been encountered at the time of the second operation. Multiple

FIG. 2-A

FIG. 2-B

Fig. 2-A: In the author's second case, lengthening was obtained by Magnuson's method, with skeletal traction applied at the time of operation. The length was increased one and one-half inches during a period of twenty to thirty minutes, and the fragments were held by transfixing screws.

Fig. 2-B: Gangrene necessitated amputation through the osteotomy site after eight days. This roentgenogram shows osteomyelitis and a sequestrum in the stump after four months. The sequestrum was removed at that time.

long incisions were made immediately in the skin, fascia, and muscles of the foot, ankle, leg, and lower thigh. Massive doses of gas-bacillus antitoxin were used, with repeated blood transfusions and large amounts of sulfonamides. (Penicillin was not available at that time.) The gas spread rapidly throughout the tissues of the thigh and about the entire hip, and extended upward over the abdomen to cover the lower portion of the thoracic cage. Multiple long incisions were made repeatedly over the involved areas, as the gas continued to spread. Further spread of the gas then ceased and the patient's general condition began to improve. A definite line of demarcation gradually appeared at the level of the osteotomy site, and the extremity distal to this point became completely gangrenous.

On the eighth day, a guillotine type of amputation was performed through the site of the original osteotomy. Osteomyelitis developed in the proximal fragment (Fig. 2-B), and a revision of the stump with excision of a sequestrum was carried out four months later. A satisfactory stump was obtained, and a prosthesis has been used without difficulty.

The next step in this chain of events was the use of a supracondylar Z-shaped osteotomy; a longitudinal slot, cut in the outer fragment, permitted it to slide along two screws, inserted loosely through the slot into the opposite fragment (Fig. 3). In this manner apposition of the two fragments was maintained.* An increase in length of two inches was obtained as planned, but the added surgery, trauma, and removal of bone resulted in delayed union. Union, in fact, was extremely poor at seven months, and a fracture occurred as a result of a trivial injury. At the end of ten months the screws were removed and the area was reinforced with iliac-bone grafts, after which fairly satisfactory union was obtained.

It was reasoned that, if control of the fragments could be maintained in this fashion, the same principle could be followed in a subtrochanteric Z-shaped osteotomy, a slot in a

* Credit for this idea belongs to John R. Schwartzmann, Resident in the Hospital, and the operation was performed with him.

commercial blade-plate (Fig. 4) being used for fixation and control of the fragments. On April 4, 1946, the first operation of this type was performed. The only difficulty encountered was in cutting smoothly the posterior cortex. The reciprocator mechanism on a Luck saw would not effect the cut, and it was completed with a thin osteotome. An increase in length of two inches was obtained in three weeks, with skeletal traction of seventy pounds in an ordinary Thomas splint. Alignment and apposition of the fragments were maintained satisfactorily (Figs. 5-A, 5-B, and 5-C).

Three operations have now been performed in this manner. For the second one a circular-saw blade was prepared, three inches in diameter, with which a longitudinal cut through the posterior cortex could be made smoothly and easily. Again an increase of two inches in length was obtained over a period of three weeks, with skeletal traction of seventy pounds.

In the third case, in which shortening of two inches had resulted from a disturbance of growth due to osteochondromata, the hip joint was normal. (In the two previous cases, the hip had been stiff.) The mobility of the proximal fragment increased the operative difficulty. The blade of an ordinary blade-plate was apparently too wide for the narrow cavity of the femoral neck. After the blade had been partially inserted, the impingement made it impossible either to insert it farther or to extract it. One and one-half hours of hard work followed before the blade was finally removed; considerable damage was inflicted upon the articular cartilage of the hip, and it appeared hemorrhagic in areas. The operation was then completed with a much shorter blade. Traction of ninety pounds was required to gain two inches in length during a three-week period.

The traction must be maintained for approximately twelve weeks before sufficient callus forms to ensure maintenance of length after a plaster cast has been applied. Roentgenographic examination is repeated at intervals of two weeks during this period.

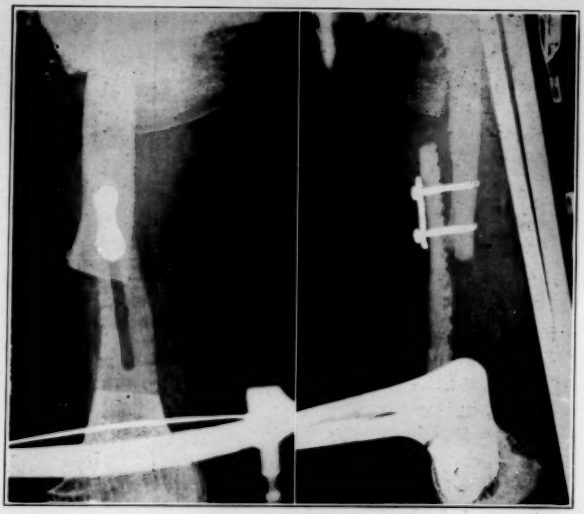

FIG. 3

Supracondylar osteotomy with a slot cut in the distal fragment to serve as a guide as lengthening proceeded. (Method devised by John R. Schwartzmann, M.D.) Increase in length was obtained, but delayed union and fracture followed.

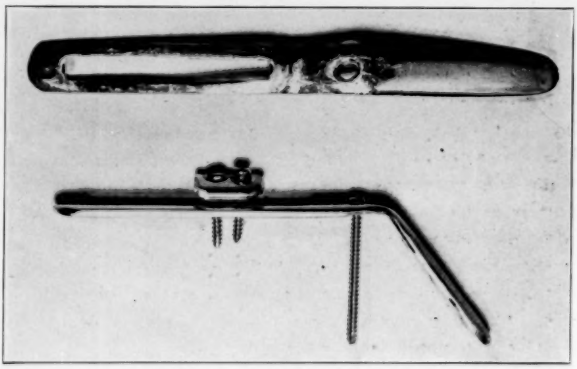

FIG. 4

Slotted blade-plate used for control of fragments as lengthening develops after subtrochanteric Z-type of osteotomy. This was used in author's last three cases.

FIG. 5-A

FIG. 5-B

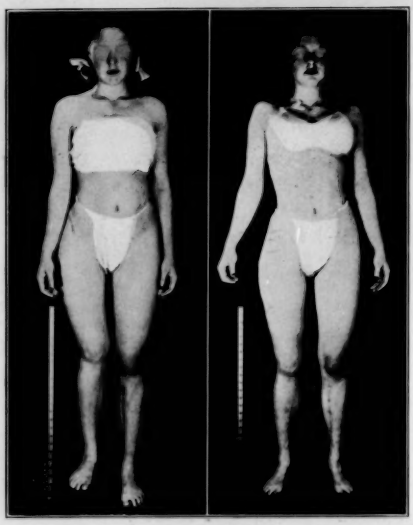

FIG. 5-C

Fig. 5-A: Shows maintenance of alignment and beginning union after four and one-half months, in the first case in which lengthening was achieved by the slotted blade-plate method.

Fig. 5-B: Degree of union after two and one-half years. The blade-plate was removed after one year and the site of osteotomy was reinforced with iliac-bone grafts.

Fig. 5-C: Preoperative and postoperative photographs show gain in length of two inches.

The roentgenogram taken at eight weeks showed the hip in a satisfactory position (Fig. 6-A). The one at ten weeks did not adequately include the hip but, since the osteotomy was well visualized, the film was not repeated. At twelve weeks the roentgenogram revealed, much to our surprise, a complete inferior dislocation of the hip (Fig. 6-B). Traction was immediately removed and a plaster spica was applied after the dislocation had been easily reduced. In view of the trauma inflicted on the femoral head at the time of operation, and a subsequent dislocation, we feared that aseptic necrosis of the femoral head might result. After one year, there was some flattening and irregularity of the head (Fig. 6-C), even though the hip had been continuously protected from weight-bearing.

A study of this series of cases would indicate that a subtrochanteric Z-type of osteotomy with the use of a slotted blade-plate, for control of the fragments as length is increased, is the most satisfactory method known at present for femoral lengthening. The details of the surgical procedure and the all-important postoperative care may be described briefly as follows:

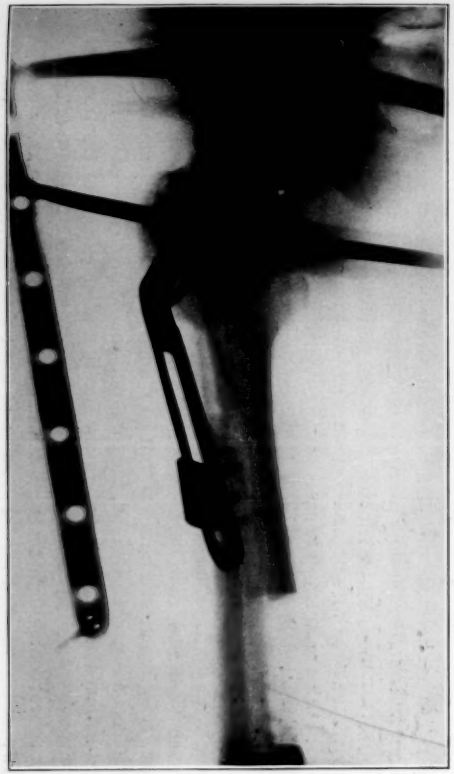

FIG. 6-A

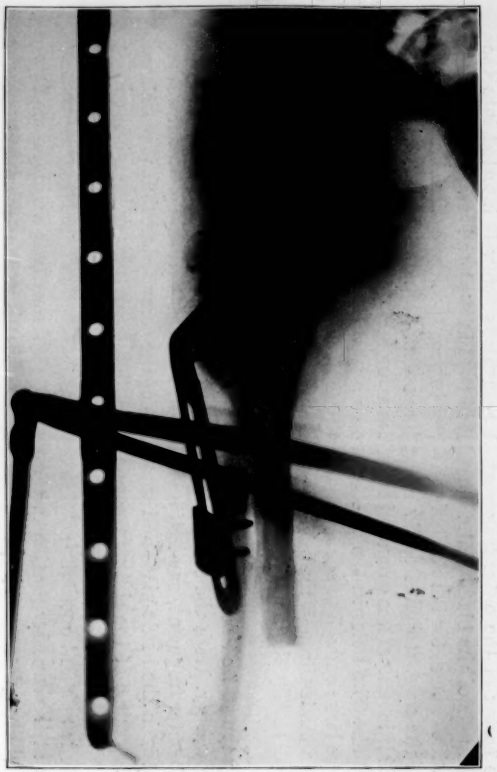

FIG. 6-B

FIG. 6-C

Fig. 6-A: In the third case in which the slotted blade-plate was used, the hip on the affected side was normal. A gain in length of one and three-quarters inches was attained. This shows evidence of early callus formation and beginning subluxation of hip, eight weeks after operation, during which time skeletal traction had been maintained.

Fig. 6-B: Complete inferior dislocation of hip was noted at twelve weeks. Traction was removed, the dislocation was reduced, and plaster was applied.

Fig. 6-C: Satisfactory alignment and union were present after one year, but there was evidence of irregularity of the femoral head. This change probably represents early aseptic necrosis.

The hip and the entire upper third of the femur are exposed through a Smith-Petersen type of approach, which is extended distally the required distance, then curved posteriorly to afford better lateral exposure by division of the iliotibial tract. A longitudinal cut, five inches in length, is made through the mid-portion of the anterior cortex of the femur, beginning at a point just distal to the flare of the greater trochanter. An ordinary circular-saw blade is used. A larger blade, three inches in diameter, is then used to make a similar parallel cut through the posterior portion of the cortex, the blade being inserted through the anterior cut previously made. With a Gigli saw, a transverse cut is made through the medial half of the cortex at the inferior point of the longitudinal cuts. With a circular-saw blade, the outer half of the cortex is cut transversely at the superior extremity of the longitudinal cuts. A thin osteotome is used to gently free and separate the two fragments. The cut surfaces are found to be perfectly smooth and even, and the two fragments are equal in size.

The slotted blade-plate is then inserted in the usual manner until the plate comes into contact with the cortex of the outer fragment. A transfixing screw is inserted into the trochanter at the angle of the blade-plate. The slot in the blade-plate is two and one-half inches long and wide enough for the head of the screw to pass through easily. This amount of width permits the screw to glide easily along its course. A metal washer, containing screw holes one-half inch apart, is prepared from the same metal of which the blade-plate and screws are made. The holes in the washer are comparable to the screw holes in any bone plate, and are large enough to accommodate the shaft of the screw, but not the head. A drill hole is then made through the outer fragment resulting from the osteotomy, at the upper point of the slot in the blade-plate. A three-quarter-inch screw is placed through one hole of the washer and is anchored into this drill hole in the outer fragment. It is allowed to engage the entire thickness of the cortex of the outer fragment, but is not tightened against the washer. With the second hole of the washer as a guide, a second screw is attached to the outer fragment, one-half inch distal to the first screw. The metal washer remains loose between the blade-plate and the heads of the screws, and thus is permitted to glide along the course of the slot as traction is applied later. This mechanism can be demonstrated with momentary manual traction at the time of operation. Since the slot in the blade-plate is two and one-half inches long, and the screws are one-half inch apart and are inserted at the superior portion of the slot, it is obvious that an increase of two inches in length will have been obtained when the screws come into contact with the lower pole of the slot. If a greater increase in length is desired, a blade-plate with a longer slot can be used.

After the operation has been completed, a threaded wire is inserted through the lower portion of the femur and another through the upper portion of the tibia, and traction bows are added. Two wires are used, for division of the strong pull and for maintenance of the traction in the event that one wire breaks. The patient is returned to bed, the lower extremity is suspended in an ordinary Thomas splint, and traction of forty pounds is added. The foot of the bed is elevated, so that the body weight can be used for countertraction. After about five days, the traction is increased to sixty pounds. Repeated portable roentgenograms are obtained and the traction is increased as necessary, in order to ensure that the entire increase in length is accomplished within a period of three weeks. Once the desired length has been obtained, the two screws in the slot of the blade-plate will prevent a further increase. It is important, however, not to reduce the amount of traction after the desired length has been gained. Instead, the total pull should be continued until roentgenograms reveal sufficient callus to maintain the position after release of traction and application of a plaster spica. This usually requires twelve weeks from the date of operation. During this time, roentgenograms are obtained at intervals of two weeks.

The plaster spica is continued for six months, or until there is evidence of adequate bony union. Gradual weight-bearing with the aid of crutches is then permitted. The plate

was removed in each of the three cases at the end of one year. In two patients, the osteotomy site was reinforced with cancellous bone from the corresponding ilium at the time the plate was removed.

It is obvious that the operation for femoral lengthening is a serious undertaking. The possible complications are great, both in number and magnitude. Prolonged hospitalization is required, constant attention to detail, and at least one year of the patient's life. All of these factors should be seriously considered and discussed with the patient before a final decision is made. If any simpler alternative measures can be utilized with anticipation of satisfactory results, or if the patient is willing to use a permanent lift on the shoe, femoral lengthening should not be attempted. Perhaps the difficulties and complications are so great that we should place the operation in complete discard; certainly before it is considered, several requirements must be evaluated carefully. These may be listed briefly as follows:

1. Shortening of one lower extremity of at least one and one-half inches must be present.
2. The individual must be too old for epiphyseal arrest and yet not past the young-adult stage.
3. The individual's stature must be so short that femoral shortening is wholly undesirable.
4. The shortening must be located principally or completely above the knee, rather than below.
5. The hip on the involved side must be stiff.
6. The extremity must be brace-free as regards the knee and leg.
7. The individual must be one who is unwilling to maintain a permanent lift on the shoe.
8. The individual's temperament and make-up must be such that one could expect him to endure the hardships of such surgery without undesirable emotional complications resulting.

An attempt has been made to analyze this series of thirty-seven femoral lengthenings in such a way as to produce an appreciation of the trials and tribulations involved. Many different methods of performing the operation have been utilized, and no single one is free from possible complications or even from catastrophe. These complications are so numerous and the prerequisites are so exacting that few patients can be classified as suitable for this procedure. For that rare individual, however, who presents all the necessary prerequisites and is willing to endure the required time, discomfort, and inconvenience, the use of the slotted blade-plate seems, at present, to afford a relatively safe and accurate method of performing the operation for femoral lengthening.

NOTE: The author wishes to express his thanks to LeRoy C. Abbott, M.D., and to C. H. Crego, M.D., for permission to study their cases, and to pay tribute to Dr. Abbott, who performed twenty-six of the thirty-seven operations reported here. One cannot study this series of cases without great admiration for the ingenuity, convictions, and surgical ability of a man who did such pioneer work almost a quarter of a century ago. All credit for the development of the operation in this country and its eventual establishment on a sound scientific basis should be given Dr. Abbott.

REFERENCES

1. ABBOTT, L. C.: The Operative Lengthening of the Tibia and Fibula. J. Bone and Joint Surg., **9**: 128–152, Jan. 1927.
2. ABBOTT, L. C.: Lengthening of the Lower Extremities. California and West. Med., **36**: 6–12, 1932.
3. ABBOTT, L. C., and CREGO, C. H.: Operative Lengthening of the Femur. Southern Med. J., **21**: 823–831, 1928.
4. BLOUNT, W. P., and CLARKE, G. R.: Control of Bone Growth by Epiphyseal Stapling. A Preliminary Report. J. Bone and Joint Surg., **31-A**: 464–478, July 1949.
5. BOST, F. C.: The Operative Lengthening of the Bones of the Lower Extremity. *In* Lectures on Reconstruction Surgery, American Academy of Orthopaedic Surgeons, pp. 50–58. Ann Arbor, Michigan, Edwards Brothers, Inc., 1944.

6. CODIVILLA, A.: On the Means of Lengthening, in the Lower Limbs, the Muscles and Tissues which are Shortened through Deformity. Am. J. Orthop. Surg., **2**: 353–369, Apr. 1905.
7. COMPERE, E. L.: Indications for and against the Leg-Lengthening Operation. Use of the Tibial Bone Graft as a Factor in Preventing Delayed Union, Non-Union, or Late Fracture. J. Bone and Joint Surg., **18**: 692–705, July 1936.
8. FREIBERG, A. H.: Codivilla's Method of Lengthening the Lower Extremities. Surg., Gynec., and Obstet., **14**: 614–617, 1912.
9. HAAS, S. L.: Lengthening of the Femur with Simultaneous Correction of Coxa Vara. J. Bone and Joint Surg., **15**: 219–224, Jan. 1933.
10. HAAS, S. L.: Retardation of Bone Growth by a Wire Loop. J. Bone and Joint Surg., **27**: 25–33, Jan. 1945.
11. MAGNUSON, P. B.: Lengthening Shortened Bones of the Leg by Operation. Ivory Screws with Removable Heads as a Means of Holding the Two Bone Fragments. Surg., Gynec., and Obstet., **17**: 63–71, 1913.
12. MAGNUSON, P. B.: Discussion of paper by Vittorio Putti. J. Am. Med. Assn., **77**: 935, 1921.
13. PHEMISTER, D. B.: Operative Arrestment of Longitudinal Growth of Bones in the Treatment of Deformities. J. Bone and Joint Surg., **15**: 1–15, Jan. 1933.
14. PUTTI, VITTORIO: The Operative Lengthening of the Femur. J. Am. Med. Assn., **77**: 934–935, 1921.

DISCUSSION

DR. LEROY C. ABBOTT, SAN FRANCISCO, CALIFORNIA: I am very grateful to Dr. McCarroll for the privilege of discussing his excellent paper, in which he has traced the development of operative lengthening of the femur at the Shriners' Hospital for Crippled Children in St. Louis.

Over a period of seven years, a group of us performed bone-lengthening operations on the lower extremities of seventy-three patients; forty-seven had lengthening of the tibia and fibula, and twenty-six had lengthening of the femur. I believe that lengthening was secured in enough cases of this large series to make the procedure worth while, for all of the surgeons at the Shriners' Hospital at that time were enthusiastic about bone lengthening. Furthermore, the operations were carried out at the same time, in one Hospital, and under conditions which were ideal for a procedure of such great magnitude.

In this paper Dr. McCarroll has made important contributions to the subject of bone lengthening. His analysis of the cases was made from a purely objective standpoint, and he concluded that operative lengthening of the femur is a highly technical and hazardous undertaking, fraught with the danger of serious complications. It should not be attempted by the inexperienced surgeon. In addition, he showed that the procedure can be simplified and the desired length can be obtained by skeletal traction on the lower fragment, with countertraction secured by the body weight of the patient. This is a progressive step, because it reduces considerably the hazards of the operation. As Dr. McCarroll has demonstrated, the ideal case is one where the hip is ankylosed. With his method, control of the position of the fragments is secured by a slotted plate. In my opinion, adequate control might be obtained by an intramedullary pin.

A further contribution made by Dr. McCarroll is the application of traction above and below the knee. This minimizes the pressure on the cartilaginous surfaces of the joint and, therefore, the chances of occurrence of a stiff knee,—a complication which is resistant to all forms of treatment.

I am interested in Dr. McCarroll's method because he has not found it necessary to do extensive division of the soft parts. I have always felt that this step, together with adequate apparatus for control of the soft parts, is an important part of the operation. In this respect I think Dr. Frederic Bost's procedure is the best because, as far as I am aware, his results show better control of the fragments.

As I have stated on numerous occasions, the procedure of limb lengthening has yielded valuable allied information. From experience with the operation, we have learned the points of fixation of important blood vessels and nerves in the lower extremity,—knowledge which is valuable in operative treatment of lesions of these structures. Furthermore, principles of limb lengthening are applicable to the treatment of complicated fractures, both open and closed, and to the treatment of old fractures in which angulation of the fragments has produced a shortening in the bone. Some of the more popular apparatus used for treatment of fractures by multiple-pin fixation are nothing more or less than modifications of the apparatus for limb lengthening.

ENDOSTEAL-BONE FORMATION IN ESTROGEN-TREATED MICE *†

BY MARSHALL R. URIST, M.D., ANN M. BUDY, B.S., AND
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The skeletal reaction of the mouse treated with large doses of estrogen is the most important recent discovery in bone physiology. Within ten years after Kyes and Potter and Gardner [6] had reported a few simple observations in birds and mice, estrogenic hormones became widely used for the treatment of healing fractures, osteoporosis, osteogenesis imperfecta, and various other types of rarefying disease of bone. The clinical results have frequently been uncertain, and suggest the need for further experimental study of estrogen treatment in bone disease.

The mode of action of the hormone on bone tissue is not known. The significance of endosteal-bone formation in the mouse and its possible relationship to the metabolic reactions of estrogen-treated patients is frequently cited in clinical reports, but actually is not quite clear. The bone changes in mice *resemble* the intramedullary bone formation which occurs in birds during the preovulatory phase of the egg-laying cycle.

The theory that estrogen stimulates osteogenesis faces a serious obstacle when experiments are transferred from birds to mammals. Many species of mammals [16, 27, 29, 30] and the human [28], when treated with large doses of estrogen, have shown only indirect effects or relatively slight changes in calcium retention and no direct evidence of physiological stimulation of osteogenesis. Nothing comparable to the striking endosteal reaction of the mouse has been produced in any other species of laboratory mammals. The mouse bone reaction, however, is a new experimental tool with which to study the circumstances and the stimuli which produce new bone tissue. Observations upon the gross, microscopic, roentgenographic, pharmacological, and endocrine features of the skeletal reaction of the mouse will be described here in detail. Evidence will be submitted which suggests that the estrogen-induced bone changes are a simple reaction to injury of the osteogenetic connective tissues of the primitive marrow.

MATERIALS AND METHODS

White laboratory mice of the CFI, IF, or CHI strain, fed on a diet of "Lab Chow Checkers", were used in all of the experiments, unless otherwise stated. The ages of the animals, the treatment, and the methods of observation will be presented in an account of the experiments and results. Histological sections of the bones and soft tissues were made by techniques described in a previous report by Urist and McLean. Serum calcium was determined by the method of Clark and Collip, serum phosphorus by the method of Gomori, and serum alkaline phosphatase by the method of Bodansky. The liver, spleen, and lymph nodes were also examined microscopically, in order to compare the soft-tissue and bone-tissue changes in the same animal.

THE HISTOGENESIS OF ENDOSTEAL NEW BONE

Experimental Procedure

Sixty-five mice, weighing from 18 to 30 grams, were injected with from 0.5 to 2.0 milligrams of α-estradiol benzoate. The animals were autopsied twenty-four hours after

* These investigations were aided by grants from the Division of Research Grants and Fellowships of the National Institute of Health, United States Public Health Service; from the Josiah Macy, Jr. Foundation; and from the Wallace C. and Clara A. Abbott Memorial Fund of the University of Chicago.

† Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 26, 1949. A preliminary communication was published in the *Proceedings of the Society for Experimental Biology and Medicine* [30].

the last dose, throughout a period of from one to fourteen days. The bones were examined by both roentgenographic and histological methods.

Results

The first changes in the skeleton were noted in the lower end of the femur and the upper end of the tibia at seven to nine days in 30 per cent. of the treated animals. At ten to fifteen days, 100 per cent. of the treated mice, but none of the controls, showed bone reactions. After fifteen days and treatment with 2.0 milligrams of α-estradiol benzoate, the spongiosa grew in size and density until it was twice the normal length and nearly five times the density. In the bird, as stated by Bloom and his associates, new-bone formation under the influence of estrogen arises from the endosteum lining the shafts of the long bones, and advances concentrically toward the axis of the shaft, following the vascular pattern of the bone marrow as it advances, until finally the marrow space is filled with new bone. Contrary to this picture, new bone is first laid down in the mouse by apposition on the trabeculae of the spongiosa at both ends of the long bones, and then advances until the marrow cavity is filled with new bone, the formation of new bone involving the endosteum of the shaft as it advances. The bone encroaches on the vascular areas of the marrow, completely disrupting the pattern and organization; small areas of bone marrow, however, remain between the spicules of bone. The process of new-bone formation in the mouse, at least in its early stages, resembles more closely that seen during recovery from the effects of large doses of parathyroid extract in the rat [18] than it does the effects of estrogens in the bird. In proportion to the rapid bone formation, osteoclastic activity appears to be markedly decreased (Figs. 1-A, 1-B, 2-A, 2-B, 2-C, and 2-D). As in the bird, new osteoblasts appear to develop by transformation of the reticular or primitive connective-tissue cells of the bone-marrow stroma.

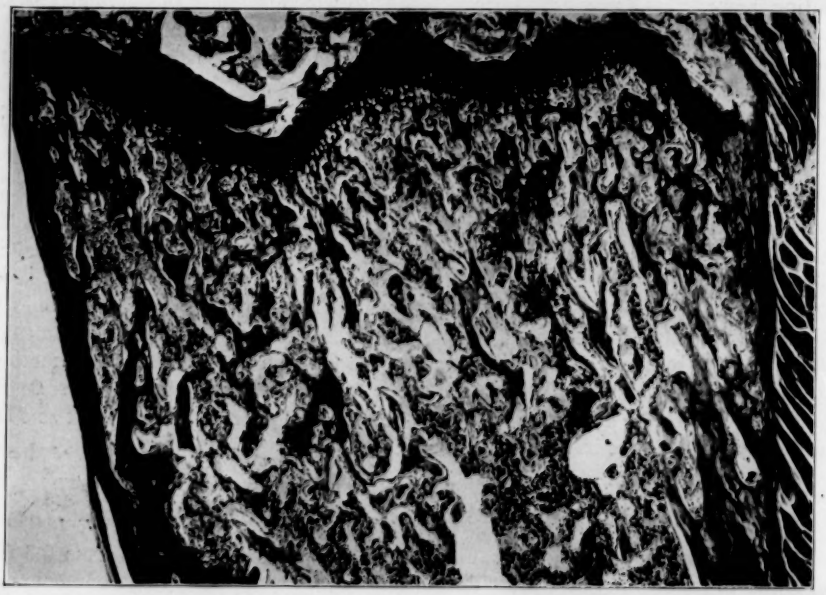


FIG. 1-A

Photomicrograph of the distal end of the femur in a forty-day-old mouse, ten days after treatment with 2.0 milligrams of α-estradiol benzoate. The spongiosa is wider and denser than the normal picture, shown in Figs. 2-A to 2-D. The new endosteal bone consists of amorphous branches of new trabeculae between the old spicules. A certain amount of the new bone contains cores of unabsorbed cartilage matrix, indicating retardation of the normal process of bone resorption at the epiphyseal lines.

THE USE OF THE MOUSE FEMUR AS A METHOD OF MEASURING THE OSTEOGENETIC REACTION OF THE SKELETON

By means of combined roentgenographic and histological techniques, it was observed that endosteal new-bone formation began precipitously and proceeded more rapidly in the femur than in any other part of the skeleton. The total reaction from the first change to complete obliteration of the medullary cavity of a femur could be sharply divided into four stages. As shown in Figures 1-A, 1-B, 2-A, 2-B, 2-C, and 2-D, and 3, these four stages were indicated on a + to + + + + scale: + indicates increased density of a normal or slightly elongated metaphysis; + +, a twofold increase in the length of the distal metaphysis; + + +, replacement of all but a central portion of the marrow

FIG. 1-B

Roentgenogram of the distal end of the same femur shown in Fig. 3. This is the earliest stage of the endosteal process of new-bone formation in the estrogen-treated mouse and is hardly evident in the roentgenograms, except that there is an increase in the length of the metaphysis as compared with the normal (Fig. 3).

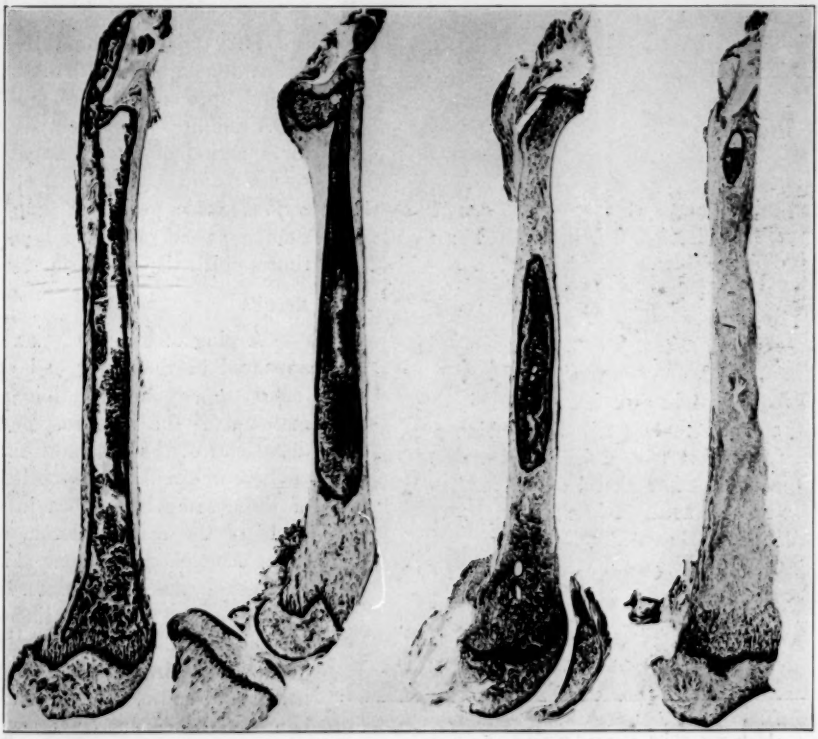


FIG. 2-A FIG. 2-B FIG. 2-C FIG. 2-D

Retouched photomicrographs of the femora of litter-mate mice, treated with large doses of α-estradiol benzoate in corn oil.

Fig. 2-A: Control injected with 0.20 milliliter of corn oil in eight weeks.

Fig. 2-B: 2.0 milligrams in two weeks.

Fig. 2-C: 8.0 milligrams in four weeks.

Fig. 2-D: 8.0 milligrams in eight weeks. Note the replacement of the marrow cavity by endosteal-bone formation.

cavity in the mid-shaft of the femur by endosteal bone growing in from both metaphyses; and + + + +, complete replacement of the entire primary marrow cavity. Routine roentgenograms alone were sufficient to show + + to + + + + reactions. Histological sections were frequently desirable, in addition, to identify a + reaction (Figs. 2-A, 2-B, 2-C, 2-D, and 3).

The quantitative results described in the following experiments could be repeated consistently, and are based upon examination of hundreds of mice. The method was very accurate when the mice were selected carefully and the test conditions were uniform. An animal of CFI strain, aged thirty days, weighing 16 to 20 grams, weaned to a "Lab Chow" diet, was considered the standard mouse. Runts, diseased mice, or otherwise defective animals, which did not grow at a uniform rate, showed irregular, erratic, or retarded responses and were discarded.

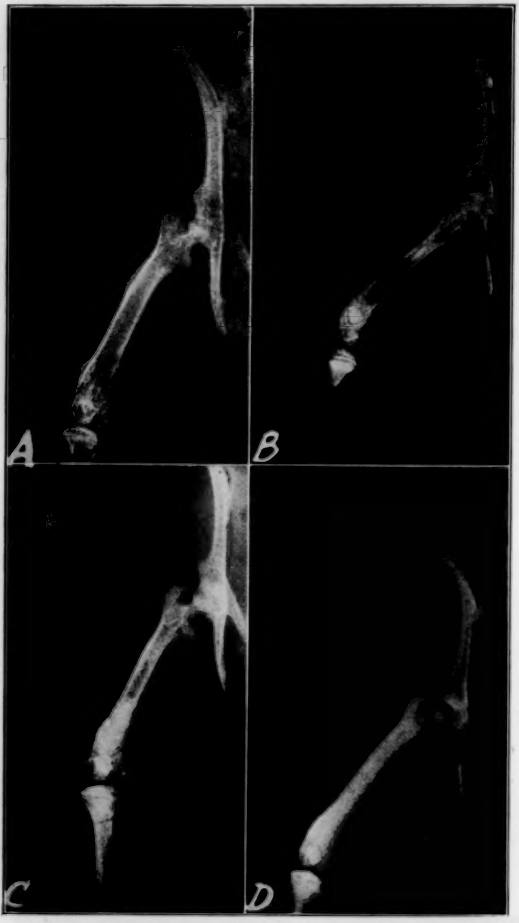


FIG. 3

Roentgenograms of the femora of litter-mate mice treated with large doses of diethylstilbestrol in corn oil.
A: Control injected with 0.4 milliliter of corn oil.
B: 6.0 milligrams in five weeks.
C: 14.0 milligrams in fifteen weeks.
D: 16.0 milligrams in seventeen weeks.
The volume and distribution of the new bone can be determined as accurately by roentgenograms as by histological sections.

THE ENDOSTEAL REACTION OF DIFFERENT PARTS OF THE SKELETON

Experimental Procedure

Fifty mice weighing 16 to 20 grams were injected with diethylstilbestrol, approximately 1.0 milligram in 0.1 milliliter of corn oil per week, for a period of one to twenty-five weeks. Animals were sacrificed when serious toxic reactions appeared, such as excessive weight loss, diarrhoea, epistaxis, or anuria.

Results

A plug of endosteal new bone appeared in the lower end of the femur, upper end of the tibia, proximal end of the humerus, and the distal end of the radius at the end of three weeks. At the opposite ends of these same bones and in other parts of the axial skeleton, either slight or no changes appeared in the roentgenograms at this time. Histologically, there was a thin line of the typical "purple-staining" estrogen-formed bone in some parts of the pelvis and spine, but the cuboidal bones of the carpus or tarsus were perfectly normal (Fig. 4-E). Between ten and fifteen weeks of treatment, the long bones were completely filled with endosteal bone and the roentgenograms showed striking increase in density of the spine and flat

bones. Slight deposits of new bone were found in the cuboidal bones in histological sections; the deposits increased in density and volume sufficiently to be seen in the roentgenograms after fifteen weeks (Fig. 5). The pubic symphysis was resorbed and appeared greatly widened from the time of the first appearance of endosteal new-bone formation.

The pattern, sequence, and rate of the bone changes in estrogen-treated mice are similar to those of the growth-rate differential of the skeleton. Although estrogen-stimulated endosteal-bone formation is a distinct reaction, separate from normal growth, the new deposits were largest in the points in the skeleton which account for most of the increase in length of the bones during the period of growth.

PATHOLOGICAL CHANGES IN THE SOFT TISSUES OF ESTROGEN-TREATED MICE

The experiments with mice treated with diethylstilbestrol, just described, showed that estrogen produces degenerative changes in the osteogenic connective tissue. It can be shown that changes also occur in the blood-forming and blood-destroying tissues, and in the cellular elements of the connective tissue itself. The limits of the subject matter and the space allotted to this paper do not permit a detailed description of the histopathological lesions, but a few of the obvious changes must be mentioned.

The blood-forming tissues are of particular interest. Ectopic hematopoiesis in the mouse occurs normally in the spleen, and, to a lesser extent, in the lymph nodes, liver, and adrenals. The damage to the lymphoid and myeloid tissues in the estrogen-treated mouse is proportional to the dose and length of treatment. Ectopic activity does not increase significantly up to ten weeks of treatment. From ten to fifteen weeks there is a marked increase in activity, which tends to approach a maximum between fifteen and twenty weeks. After twenty weeks of continuous treatment, there is a sharp decline in hematopoietic activity. The blood-forming tissue is replaced by fibrous connective tissue; lymph nodules are depleted; cellular elements become hypertrophied; and necrosis soon follows. The final stages at twenty-five weeks are fibrosis, hyalinization, and colloid degeneration of the lymphoid and myeloid tissues everywhere in the body. Until the very end of treatment, some hematopoietic marrow could always be found in narrow cords between the bone trabeculae in all parts of the skeleton.

THE PROCESS OF RECUPERATION

Experimental Procedure

Fifteen immature mice were injected with 0.5 milligram of α-estradiol benzoate per week for four weeks; treatment was then discontinued. All of the animals were examined by roentgenograms at intervals, and one or two were autopsied every two weeks from four to thirty weeks of recovery.

Results

The roentgenograms showed a + + reaction in all fifteen mice at four weeks of treatment. Two weeks after estrogen had been discontinued, all showed progressive endosteal-bone formation to the stage of a + + + reaction. Four weeks after treatment, only one mouse showed a + + + + reaction; all the others showed no further increase in the size of the deposits.

The normal picture of endochondral ossification, including the normal process of resorption of cartilage matrix and bone, reappeared just below the epiphyseal lines, six weeks after treatment had been discontinued. The epiphyseal cartilage had grown away from the zone of endosteal bone formed under estrogen treatment, and a radiolucent line had formed across the primary spongiosa. The epiphyseal plate continued to grow away from the endosteal deposit until it was separated from it by 1 millimeter at eight weeks and by 1.4 millimeters at twelve weeks. The density of the endosteal deposits began to diminish

FIG. 4-D

FIG. 4-C

FIG. 4-B

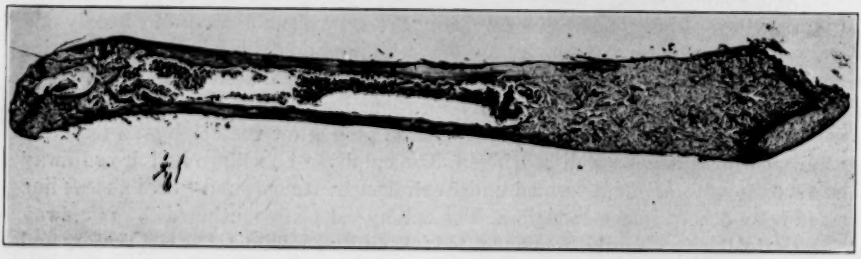


FIG. 4-A

Fig. 4-A: Photomicrograph (× 11) of the distal end of the femur in an eight-week-old mouse treated with 4.0 milligrams of diethylstilbestrol during a period of four weeks. Approximately one-half of the marrow cavity has been replaced by new bone.

Fig. 4-B: Photomicrograph (× 104) of a section through the costochondral junction of a rib of the same mouse. Some endosteal-bone formation is evident, but the process is much less advanced than in the femur.

Fig. 4-C: Photomicrograph (× 52) of a lumbar vertebra shows the same degree of change as at the costochondral junctions. Significant deposits of new bone are found mainly at the epiphyseal lines.

Fig. 4-D: Photomicrograph (× 13) showing a sagittal section through the blade of an ilium. The volume of the marrow cavity is normal and the amount of endosteal new-bone formation is insignificant. Examination of the tissues under oil-immersion high-power magnification showed a very thin layer of new bone on some of the trabeculae nearest to the acetabulum.

Fig. 4-E: Photomicrograph (× 56) of a longitudinal section through the tarsus. Except perhaps under very high-power magnification, there was no evidence of new-bone formation.

FIG. 4-E

between twelve and fourteen weeks. At sixteen weeks the estrogen-formed bone deposits were less than half the previous density; and in two mice, autopsied after eighteen weeks, the endosteal bone was almost completely absorbed (Figs. 6-A to 6-F).

THE INFLUENCE OF DEVELOPMENT, GROWTH, AGE, AND HEREDITARY STRAIN

Experimental Procedure

1. Seven foetal mice were obtained at term from five pregnant animals which had been injected with 1.0 milligram of diethylstilbestrol per week, beginning seven days after insemination. The skeletons of the mothers and of the young were examined near term for intramedullary new bone.

2. Four litters of fourteen newborn mice were injected with α-estradiol benzoate or diethylstilbestrol, beginning on the day of birth and then at seven-day intervals with graduated doses of 0.05 milligram (until fourteen days), to 0.1 milligram (until twenty-eight days), and to 0.5 milligram (after thirty-five days). Another litter was injected with olive oil and the animals were used for controls. One animal from each litter was sacrificed every five days between five and forty days of age.

3. Four groups of ten mice each, aged three, four, five, and six months, were injected with α-estradiol benzoate, 0.5 milligram per week. The animals were autopsied after four, eight, and twelve weeks of treatment.

4. Fifty mice, aged one year or more, were injected with 0.5 milligram of diethylstilbestrol, α-estradiol benzoate, or ethinyl estradiol. They were autopsied after four, eight, and ten weeks of treatment.

5. Fifteen mice, more than one year of age, were treated with subcutaneous implants of pellets of 1.0 milligram of α-estradiol benzoate.

6. Six mice of the CHI strain* (Strong A Strain, F88 generation of breeding), reported to be resistant to the skeletal effects of estrogen, were treated with injections of 0.5 milligram per week of α-estradiol benzoate in olive oil.

Results

The skeletons of mice *in utero* of pregnant mothers treated with estrogen showed no endosteal new bone in roentgenograms. Newborn mice and young mice from treated mothers showed no gross or roentgenographic changes in the bone structure.

Histological examination of the newborn mice, treated with estrogen on the day of

* Kindly provided by Dr. Leonell C. Strong of Yale University, Department of Anatomy.

FIG. 5

Litter-mate mice, twenty-one weeks of age, placed side by side on one roentgenographic film which was exposed by focusing the tube directly between them, in order that both should receive exactly the same intensity of radiation. The mouse on the left was treated with 16.5 milligrams of diethylstilbestrol for a period of seventeen and one-half weeks, and shows complete obliteration of the marrow cavities of the long bones by new bone. To a lesser extent the spine and flat bones are also increased in density. The carpal and tarsal bones show relatively little change.

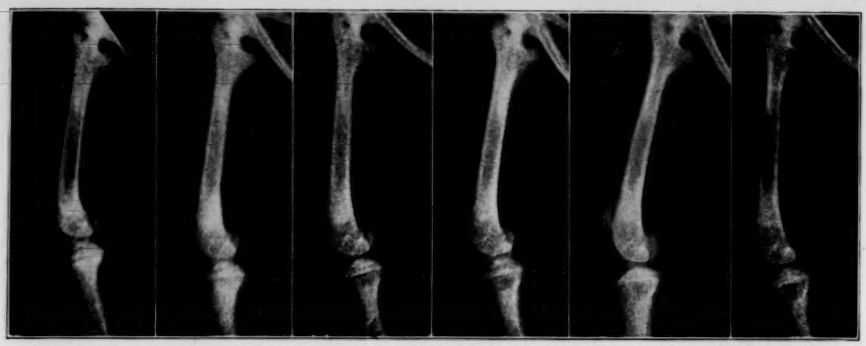


FIG. 6-A FIG. 6-B FIG. 6-C FIG. 6-D FIG. 6-E FIG. 6-F

Fig. 6-A: Roentgenogram of a knee joint of a mouse at eight weeks, four weeks after treatment with 2.0 milligrams of α-estradiol benzoate. Note the increase in density and length of the distal metaphysis of the femur and the proximal metaphysis of the tibia. At this stage of the process of endosteal-bone formation, the estrogen was discontinued. The various changes which occurred in the same region during a period of twelve weeks are shown in Figs. 6-B to 6-F.

Fig. 6-B: Four weeks after the estrogen had been discontinued, the metaphysis continued to increase in length, but a line of rarefaction appeared at the epiphyseal line.

Fig. 6-C: Six weeks after the estrogen had been discontinued, roentgenograms of the femur showed a zone, 1 millimeter in length, in which the normal process of resorption had been resumed and the bone had continued to grow. This is also evident in the upper end of the tibia.

Fig. 6-D: Eight weeks after the estrogen had been discontinued, the intramedullary deposit of endosteal bone was separated from the epiphyseal plate by a distance of approximately 1.5 millimeters.

Fig. 6-E: Ten weeks after the estrogen had been discontinued, the epiphyseal plate had grown 2 millimeters away from the endosteal bone.

Fig. 6-F: Twelve weeks after the estrogen had been discontinued, there was nearly 3 millimeters of the length of the shaft between the endosteal deposit and the distal portion of the diaphysis of the femur. The endosteal bone became reduced in density at this point, and was entirely absorbed during the period from sixteen to thirty weeks.

FIG. 7-A FIG. 7-B

Fig. 7-A: Roentgenogram (× 3) of the femur of a normal mouse, approximately one and one-half years of age. The density of the bone structure is everywhere considerably less than in a young mouse (Fig. 3). (Mice are considered aged after one year.)

Fig. 7-B: Roentgenogram of the femur of a mouse, the same age as the mouse shown in Fig. 7-A, six weeks after treatment with a total of 3.0 milligrams of α-estradiol benzoate. The amount of endosteal new bone is much less than in a similarly treated young mouse, and cystic areas appear in the metaphysis and the epiphysis.

birth, showed evidence of retarded resorption of calcified cartilage matrix as early as five days. At ten days there was an increased invasion of vascular mesenchyme eroding the calcified cartilage, accompanied by sheets of osteoblasts. The increased osteoblastic activity can be shown by the formation of primitive trabeculae before the early calcified cartilage matrix is completely resorbed. The number of unresorbed fragments scattered throughout the marrow cavity was greater than in the normal. The primitive bone trabeculae contained irregular pieces of calcified cartilage matrix. At twenty days the osteogenetic reaction to estrogen was complete enough to be detected by roentgenogram, but at earlier ages estrogen-induced bone formation could be found only by microscopic observation. Age made no difference in the response of the mice, which were from one to six months old.

Certain strains of mice live as long as two years, but breeders are generally considered aged after one year of life. Such aged mice showed some delay in the response of the skeleton to estrogen. Fifty per cent. of the animals showed negative roentgenograms until after

four weeks of treatment. When the reaction did occur after that time, the volume of endosteal bone was considerably less than that found in similarly treated young mice. The reaction of aged mice to large doses of estrogen (0.5 milligram) was quite irregular.

TABLE I

RELATIONSHIP BETWEEN THE DOSE OF ESTROGEN AND THE VOLUME OF ENDOSTEAL NEW BONE

Drug	Dose (*Milligrams*)	No. of Weeks	Reaction of the Femur
α-Estradiol benzoate	1	3	+
α-Estradiol benzoate	2	3	+ +
α-Estradiol benzoate	4	3	+ + +
Controls	0	3	0
Diethylstilbestrol	1	5	±, +
Diethylstilbestrol	2	5	+
Diethylstilbestrol	4	5	+ +
Controls	0	5	0

The first reaction was the formation of cystic areas. The endosteal bone, formed after these cystic areas had decreased, was deposited in peculiar masses, usually at the ends of the bones, but sometimes also upon the inner cortex of the mid-shaft (Figs. 7-A and 7-B). Smaller doses of estrogen, administered in the form of pellets, did not produce these bizarre deposits. In an experiment with a relatively small number of mice, it was difficult to determine how many of the aged mice were dying of incidental causes or old age, but it was apparent among the survivors that senile mice did not tolerate large doses of estrogen as well as did immature mice. In the aged mice which survived eight weeks of treatment, the usual deposits of endosteal new-bone formation eventually were seen. The sequence of the reaction in the various parts of the skeleton was the same in old mice as in the immature animals. Serial roentgenograms showed replacement of the marrow cavity at the distal end of the femur and the proximal end of the tibia by new bone when other parts of the skeleton were as yet unchanged. The spine and flat bones showed evidence of increased density much later. The cuboidal bones of the tarsus and carpus were unchanged, and contained marrow cavities until the end of the period of treatment.

There was no variation in the bone reaction in different hereditary strains. The response to estrogen was the same in the CFI and IF strains, as well as in the CHI cancer-bearing strain.

THE DOSE, VEHICLE, PERIOD OF ADMINISTRATION, AND CONTINUITY OF ACTION OF ESTROGEN, AND OTHER FACTORS INFLUENCING THE PROCESS OF ENDOSTEAL-BONE FORMATION

Experimental Procedure

1. Thirty-two mice were divided into three groups and injected one, two, and four times per week with 0.5 milligram of α-estradiol benzoate in 0.05 milliliter of corn oil. A fourth group of eight mice were injected with 0.05 milliliter of corn oil and autopsied one week after the last injection, at two, three, five, and eight weeks. The same experiment was repeated with the use of diethylstilbestrol, a synthetic estrogen.

2. Thirty mice were divided into three groups and injected with 0.5 milligram per week of diethylstilbestrol phosphate or ethinyl estradiol in: (a) corn oil, (b) normal saline solution, and (c) pellets. These estrogens were used because of their solubility in water,

TABLE II

RELATIONSHIP BETWEEN THE DURATION OF TREATMENT AND THE VOLUME OF ENDOSTEAL BONE

Drug	Dose (*Milligrams*)	No. of Weeks	Reaction of the Femur
α-Estradiol benzoate	2.0	2	+
α-Estradiol benzoate	2.0	3	+ +
α-Estradiol benzoate	2.0	5	+ + +
Controls	0	5	0
Diethylstilbestrol	2.0	2	0
Diethylstilbestrol	2.0	3	±
Diethylstilbestrol	2.0	5	+
Controls	0	5	0

in which form they were assumed to be absorbed more rapidly than from oil solution or pellets.

The pellets were made by compressing the crystalline estrogens in a cylindrical punch. They were then weighed, sterilized at 80 degrees centigrade for one hour, and implanted either subcutaneously or surgically into muscles or bones, as indicated in the experimental procedures.

Results

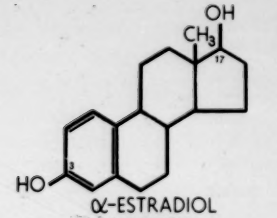
In the first three weeks of treatment with α-estradiol benzoate, or the initial period of

TABLE III

THE OSTEOGENETIC ACTION OF VARIOUS ESTROGENS IN DOSES OF 0.5 MILLIGRAM PER WEEK

Hormone	Reaction of the Femur		
	4 Weeks	8 Weeks	12 Weeks
Natural Estrogens and Chemical Derivatives of Hormones			
α-Estradiol	+ +	+ + +	+ + +
α-Estradiol benzoate	+ +	+ + +	+ + + +
α-Estradiol dipropionate	+ +	+ + +	
β-Estradiol	0	+	0
β-Estradiol benzoate*	+ +	+ +	+ + + +
Ethinyl estradiol	+	+ +	
Estrone	+	+ +	+ + +
Sodium estrone sulphate	+	+ +	+ +
Estriol	+	+	+ +
Premarin	0	0	0
Methyl dehydrodoisynolic acid	0	0	0
Synthetic Estrogens			
Diethylstilbestrol	+	+ +	+ + +
Diethylstilbestrol phosphate	+ +	+ + +	+ + +
Hexestrol	0	+	
Dienestrol	0	+	+ +

* Crude β-estradiol benzoate was recrystallized by Dr. Konrad E. Bloch of the Department of Biochemistry. The product obtained had a melting point of 152 to 153 degrees, and contained less than 1 per cent. of the original α-estradiol benzoate present.



α-ESTRADIOL
M.W.=272

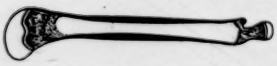
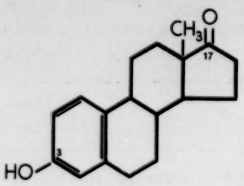
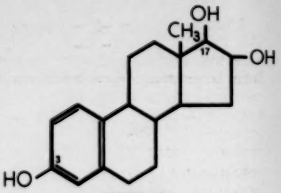


FIG. 8-A



ESTRONE
M.W.=270

FIG. 8-B



ESTRIOL
M.W.=288

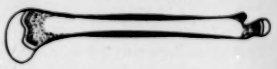
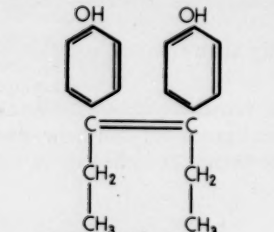


FIG. 8-C



DIETHYLSTILBESTROL
M.W.=314

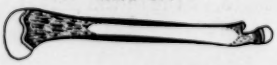
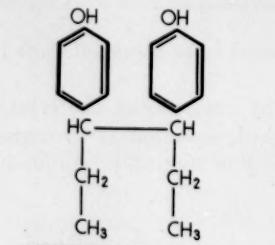
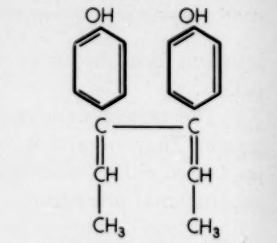


FIG. 8-D



HEXESTROL M.W.=316

FIG. 8-E



DIENESTROL M.W.=310

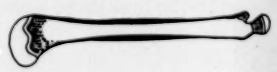
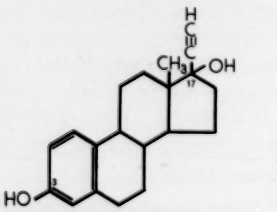
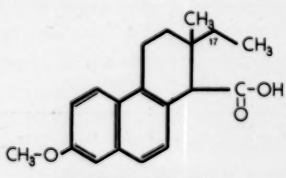


FIG. 8-F



ETHINYL ESTRADIOL MW=321

FIG. 8-G



3 METHYL ETHER
BIS-DEHYDRO-DOISYNOLIC ACID
M.W.=310

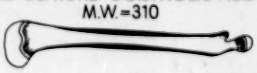


FIG. 8-H

Diagrams showing the structural formulae of eight different estrogens and the average yield of endosteal new bone in the femur obtained from each substance, given to a thirty-day-old mouse in a dose of 0.5 milligram per week for a period of eight weeks. The shaded lines indicate the size of the metaphysis and the area of the new bone. The white area in the mid-shaft indicates the corresponding decrease in the volume of the marrow cavity. The normal picture is shown in Fig. 8-H after treatment with 3-methyl-ether-bis-dehydrodoisynolic acid, which has no effect on the bones of mice.

Figs. 8-A, 8-B, 8-D, and 8-G show the maximum reaction that was obtained with high-potency unbenzoated estrogens. Figs. 8-C, 8-E, and 8-F show the relatively slight effects of low-potency estrogens.

five weeks of treatment with diethylstilbestrol, the volume of new bone deposited in the femur was directly proportional to the magnitude of the dose (Table I). After these periods of time, many of the animals treated with 0.5 milligram per week showed as much new bone as those which had received as high as 2.0 milligrams per week. The maximum response in both rate and volume of endosteal new-bone formation appeared with a 1.0 milligram dose. This was also the minimum lethal dose for 50 per cent. of the animals after four weeks of treatment with diethylstilbestrol.

After injection of a total dose of 2.0 milligrams of either α-estradiol benzoate or diethylstilbestrol in oil, within various periods of time, such as two, three, and five weeks, it was apparent that the volume of endosteal bone produced by the femur was increased

Figs. 9-A, 9-B, 9-C, and 9-D

Diagrams showing the closely related structural formulae, the average uterine reaction, and the yields of endosteal bone from biological equivalents of 4,500 international units of unesterified and benzoate esters of the alpha and beta isomers of estradiol. Benzoate esterification increased both the estrogenic and the osteogenetic effects of the female sex hormone. Esterification also conferred bone-forming action upon β-estradiol, one of the weakest of the steroid hormones.

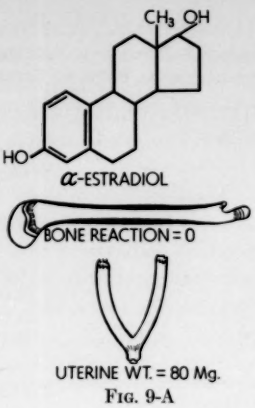


FIG. 9-A

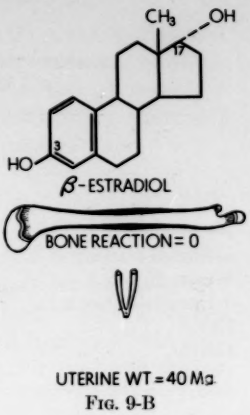


FIG. 9-B

with time or the duration of treatment (Table II).

With injections of a dose of 0.5 milligram or more of estrogen, there was no difference in the amount of endosteal bone produced within eight weeks when the drug was rapidly absorbed from saline solutions, slowly absorbed from oil solutions, or even very slowly dissolved from the surface of pellets. These results may be explained by the fact that the recovery period from the effects of these large doses of estrogen is from four to six weeks, which is much longer than the interval between the injections.

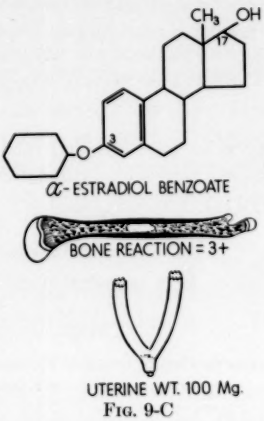


FIG. 9-C

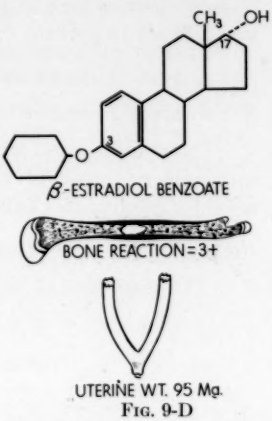


FIG. 9-D

THE LEVELS OF SERUM CALCIUM, INORGANIC PHOSPHORUS, AND ALKALINE PHOSPHATASE DURING ENDOSTEAL NEW-BONE FORMATION

Experimental Procedure

Two hundred and forty immature mice were divided into six groups. One group of thirty were injected with 2.0 milligrams of α-estradiol benzoate over a period of nine days and were sacrificed on the tenth day. Two groups of sixty each were injected with 1.0 milligram per week of α-estradiol benzoate and sacrificed after four weeks of treatment. Three groups of thirty each were similarly injected with olive oil for controls. The sera of each group of animals were pooled for determinations of serum calcium by the method of Clark and Collip, of serum phosphorus by the method of Gomori, and of serum alkaline phosphatase by the method of Bodansky.

Results

During the early stages of endosteal new-bone formation, the serum calcium and inorganic phosphorus were at the same level as that found in oil-treated control mice. At the time of rapid endosteal-bone formation, the serum calcium was 11.90 milligrams per 100 cubic centimeters, the serum phosphorus averaged 9.76 milligrams per 100 cubic centimeters, and the serum alkaline phosphatase was 8.15 (milligrams of phosphorus split off 100 milliliters of serum in one hour). The levels of the oil-treated control mice were 13.30, 10.18, and 11.25 milligrams per 100 cubic centimeters, respectively. The serum

TABLE IV

RELATIONSHIPS BETWEEN ESTROGENIC POTENCY AND OSTEOGENETIC ACTION OF 4,500 INTERNATIONAL UNITS OF ESTROGEN IN THREE WEEKS

Hormone	Dose (*Milligrams*)	Weight of Fresh Uterus (*Milligrams*)	Reaction of the Femur
α-Estradiol	0.036	800	0
α-Estradiol benzoate	0.072	700	+ +
β-Estradiol	1.50	400	0
β-Estradiol benzoate	3.00	750	+ +
Estriol	3.00	600	+
Estrone	0.45	800	+ +
Control		400	0

phosphorus was high in both treated and untreated animals, owing to hemolysis which was difficult to prevent in pooled mouse blood.

THE OSTEOGENETIC POTENCY OF VARIOUS ESTROGENS

Experimental Procedure

One hundred and sixty mice were divided into fifteen groups and treated with 0.5 milligram per week of various estrogenic substances, injected intramuscularly in 0.05 milliliter of olive oil (Table III).

Results

The benzoates of alpha or beta isomers of estradiol showed powerful osteogenetic

TABLE V

THE EFFECT OF OTHER STEROID HORMONES ADMINISTEREED ALONE AND IN ADDITION TO α-ESTRADIOL BENZOATE

Treatment	Dose (*Milligrams*)	Reaction of the Femur	
		4 Weeks	8 Weeks
Progesterone	0.5 per day	0	
	1.0 per day	0	0
Progesterone plus α-estradiol benzoate	1.0 per day 0.5 per week	+ +	+ + +
Desoxycorticosterone acetate	2.0 per week	0	0
Desoxycorticosterone acetate plus α-estradiol benzoate	2.0 per week 0.5 per week	+ +	+ + +
Testosterone propionate	1.25 per day	0	0
	3.00 per day	0	0
Testosterone propionate plus α-estradiol benzoate	10.4 per week 0.17 per week		+ +
	5.0 per week 0.5 per week	+ +	+ +
	10 to 40*		
	0.5 to 1.0	+ +	+ + +

* This experiment was done with the use of subcutaneous pellets. All others listed were done with solutions of olive oil.

action, and were at least twice as potent as any of the other natural estrogens (Figs. 8-A to 8-H). The order of magnitude of the effects of all the estrogens, including synthetic preparations which are now used commercially, are shown in Table III. To determine the relationship between osteogenetic and estrogenic potency, the two isomers of estradiol were administered in doses of biological equivalents of 1,500 international units.

Experimental Procedure

Thirty-six mice were divided into six groups and injected with 1,500 international units per week of one of the following: α or β–estradiol, α or β–estradiol benzoate, estrone, or estriol. It is believed that these isomers of estradiol are the female sex hormone as it exists in the liquor folliculi of the ovary. The estrogenic potency of each of these hormones was estimated by the uterine-weight method of Evans, Varney, and Koch, and was compared directly with the amount of endosteal bone that was produced in the same animals with biologically equivalent doses (Table IV).

Results

As shown in Table IV, the osteogenetic action of α–estradiol, one of the most potent of all estrogens, is relatively low and not greater than that of β–estradiol, which is one of the weakest estrogens which has been isolated. The benzoic-acid ester of each of these substances, however, shows extraordinary osteogenetic potency. The weaker estrogen, β–estradiol benzoate, is as active upon the bones as the more powerful estrogenic hormone. Hence, esterification of an estrogen is one of the most important factors in its osteogenetic potency. This may be due to slower absorption, more prolonged mobilization, or to a slower excretion of the esterified estrogen (Figs. 9-A, 9-B, 9-C, and 9-D).

THE SINGLE AND COMBINED ACTION OF OTHER STEROID HORMONES UPON ENDOSTEAL-BONE FORMATION IN TREATED AND UNTREATED IMMATURE MICE

Experimental Procedure

Seventy-five mice were divided into eight groups and treated with progesterone, desoxycorticosterone acetate, or testosterone propionate, alone and in combination with α–estradiol benzoate. Table V shows the dosages, intervals, methods of treatment, and results.

Results

Endosteal new-bone formation was not produced by the lutoid, corticoid, or testoid hormones. There was neither inhibitory nor synergistic action of progesterone, desoxycorticosterone, or testosterone upon the osteogenetic action of large doses of estrogen.

THE EFFECTS OF GONADOTROPIC AND ADRENOTROPIC FACTORS OF THE ANTERIOR PITUITARY

Experimental Procedure

Five immature mice were injected daily with 148.0 milligrams of a highly purified pregnant mare's serum gonadotropin (Antex [Ayerst], one international unit per 0.25 milligram) in aqueous solutions intramuscularly over a period of ten weeks.

Ten immature mice were injected daily with 5.0 milligrams of chorionic gonadotropin (A.P.L. [Ayerst], one international unit per 0.25 milligram) over a period of eight to ten weeks. Ten immature mice were injected daily with 5.0 milligrams of A.P.L. and 5.0 milligrams of Antex for a period of six weeks. This treatment tends to reproduce the hormone pattern of the reproductive cycle by utilizing the follicle-stimulating properties of Antex in the early phase and the luteinizing effect of A.P.L. in the later phase.

Six immature mice were given 2.5 milligrams of Antex plus 2.5 milligrams of A.P.L. daily, supplemented by weekly injections of 0.5 milligram of α–estradiol benzoate.

Three aged mice (breeders, one to one and one-half years of age) received the same treatment with all three substances.

Ten newborn mice also received injections of all three substances, but in doses of 3.0 and 4.0 milligrams per week of Antex and A.P.L., and from 0.05 to 0.30 milligram of α-estradiol benzoate.

Ten immature mice were given daily intramuscular injections of 1.0 milligram of Armour's adrenocorticotropic hormone, ACTH (potency approximately 35 per cent. of the standard), over a period of six weeks.

Results

Continuous stimulation of the ovaries by large doses of follicle-stimulating hormone for ten weeks failed to produce endosteal-bone formation. Chorionic gonadotropin and combined follicle-stimulating hormone and anterior-pituitary-like therapy similarly were without effect. The osteogenetic action of estrogen was neither enhanced nor inhibited by simultaneous treatment of the animals with these anterior-pituitary-hormone preparations.

Aged mice in which the osteogenetic response was generally delayed, and infantile mice which were refractory to the osteogenetic action of estrogen, when primed with both follicle-stimulating hormone and anterior-pituitary-like substance, showed no more endosteal-bone formation than unprimed controls.

Adrenocorticotropic hormone produced no endosteal-bone formation.

LOCAL IMPLANTATION OF FREE ESTROGENS IN BONE

Experimental Procedure

Pellets made of compressed crystals of two water-soluble and two oil-soluble estrogens were implanted surgically in drill holes in the distal metaphyses of the femora of twelve immature mice. Diethylstilbestrol phosphate and ethinyl estradiol, the water-soluble estrogens, were assumed to be more readily diffusible in the tissue fluids. Diethylstilbestrol and α-estradiol benzoate were the oil-soluble estrogens that were used, and they were assumed to be dissolved only slowly. Pellets of cholesterol were similarly implanted in three litter mates for controls. The bones were examined at intervals of eighteen, twenty-five, thirty-two, and forty-three days.

Results

All the implants of cholesterol and estrogen were encased in a shell of fibrous connective tissue and bone tissue. The skeleton showed the usual systemic bone reaction to absorption of implants of estrogen, but not to the implants of cholesterol, and there were no specific local osteogenetic effects.

DISCUSSION

The intermediary metabolism and the mode of action of the female sex hormones upon the genital tract is not yet known, and even less is understood about the nature of the effects of estrogen upon bone. All the possible endocrine-gland interrelationships, as well as vitamin and other dietary factors, have been studied with essentially negative results. The literature has been reviewed by Gardner and Pfeiffer, with a summary of their work prior to 1943. The facts presented bear repetition, along with a review of the reports which have been published since then and the results of experiments in this paper.

Hypophysectomy did not prevent the endosteal reaction of the bones [19, 26]. Gardner and Clouet, in 1945, reported that mice, made hypothyroid by a diet including thio-urea, were able to produce endosteal new bone. Various endocrine-gland preparations have been administered without much effect upon endosteal-bone formation. Silberberg and Silberberg, in 1946, found that thyroxin did not affect the estrogen-treated mouse, al-

though it did produce some effects on the maturation of cartilage cells. Anterior-pituitary growth hormone was found to cause more rapid regression of the epiphyseal plate, but it had little effect upon the endosteal reaction [23]. Treatment with testosterone was reported by Gardner and Pfeiffer, in 1938, to be the only procedure which averted the action of estrogen on the bones of the mouse. This is one important point at which the present observations are at variance with the previous literature. In proportions as high as sixty to one, testosterone did not inhibit the action of α-estradiol benzoate in our experiments. This observation leads us to question the hypothesis of antagonistic action of the sex hormones on the bones.

The authors have found no evidence that the mouse produces endosteal bone, characteristic of the effects of estrogens, under strictly physiological conditions. During pregnancy, when large amounts of estrogen are being excreted in the urine, neither the foetal nor the maternal skeleton showed roentgenographic evidence of estrogen-induced bone formation, although the reproductive tracts of both the mother and the newborn mouse do show the full effects of the female sex hormone. The skeleton of the estrogen-treated newborn mouse shows a typical reaction as early as ten days. In the experiments described, it was not possible to produce endosteal-bone formation in either immature or mature mice on the strength of the animals' own ovaries, stimulated by the injection of large doses of gonadotropic hormone over a long period of time.

Newborn mice did not produce endosteal bone as rapidly as immature mice. Aged mice also responded much less vigorously than immature mice and young adults. Aged animals showed degenerative joint changes [24] and some delay in the onset of the bone reaction, but eventually the usual deposits of new-bone tissue appeared in the long bones. Hence, although the mouse usually reaches its growth plateau at six months, it retains its ability to produce large quantities of new bone in response to estrogen. Even the pattern of the endosteal reaction is independent of growth, for it is the same in senile mice and immature mice. The growth potentials of the different parts of the skeleton, the so-called "growth differential", does persist and may govern the endosteal response at all ages throughout the life of the individual.

An inbred cancer-bearing strain of mice has been reported by Segaloff and Cahill to be resistant to the skeletal effects of estrogen. Our observations upon the same strain indicate that, if given a large dose (0.5 milligram per week), such mice will show the usual endosteal bone changes.

Both the magnitude of the bone response and the rate of endosteal-bone formation were directly proportional to the dose of the estrogenic substance and the duration of the treatment. The osteogenetic reaction was also related in some way to the estrogenic potency of the substance that was used. The uterine response, however, reaches its maximum long before the bone reaction is completed. Esterification with benzoic acid greatly increased the osteogenetic action of both of these substances, presumably due to the prolongation of the effects and the lower rate of excretion of the material.

The process of recuperation from estrogen treatment and the reformation of the marrow cavities was complete in approximately five months. Serial roentgenograms (Fig. 5) resemble the roentgenographic picture which is seen in Fairbank's cases of "marble-bone" disease during the period of remission. There is also a striking similarity in the large amounts of unabsorbed cartilage matrix in the bones of estrogen-treated mice and rats and in the human with marble-bone disease. Miller, Orr, and Pybus have described permanently increased thickness of the cortex after estrogen treatment in mice.

Diet apparently plays no role in the endosteal proliferation. Animals fed synthetic diets, low in protein or total nitrogen, were able to produce the usual reaction [9]. Although it was difficult for the authors to produce florid rickets in mice, estrogen-treated mice fed a low-phosphorus, high-calcium diet have been reported to lay down the osteoid tissue incompletely calcified, just as would be expected of untreated animals [22]. Mice fed on a

low-calcium, high-phosphorus diet showed advanced rickets and the same endosteal reaction to estrogen as normal mice. The authors have weaned young mice to diets containing as little as from 2 to 10 per cent. protein and found suppression of growth, but the deficiency did not prevent the appearance of the bone reaction to estrogen.

Roentgenographic methods as used here for estimating the extent of endosteal-bone formation, when combined with histological methods, were found to be more informative than bone-ash studies or measurements of the breaking strength [7]. At the early stages of the endosteal reaction, the new-bone formation is limited to the growing ends of the bones. Until the final stages of replacement of the marrow cavity, the breaking strength of the bones in the mid-shaft of the long bones may be the same as in normal animals. The percentage of ash of the bones [21, 32] provided some idea of the total increase in bone substance, but gave no information about the exact location of the new deposits in the skeleton as a whole.

When the marrow cavities of all the long bones became filled with a network of osseous tissue, there was still sufficient production of red cells and hemoglobin to sustain the mice in reasonably good condition. During a similar period of treatment on doses lower than those used in the experiments described here, Jacobson mentioned an anaemia of "questionable significance". In the authors' experiments with larger doses and after extensive filling of the marrow spaces of both the flat bones and the long bones, there was still adequate blood formation in the interstices of the endosteal bone and only moderate anaemia. This demonstrated the large resources in blood-forming tissue, but there were nevertheless deleterious effects upon the lymphoid and myeloid tissues. Ectopic hematopoietic activity increased after ten weeks of treatment. After twenty weeks of treatment, both the lymphoid and myeloid tissues in the liver, spleen, lymph nodes, and bone marrow showed necrosis and extensive fibrous degeneration.

In birds, intramedullary-bone formation is a physiological reaction of the skeleton, which is associated with the need for storage and rapid mobilization of large amounts of calcium. Birds and other egg-laying species possess a special chemical mechanism in the serum vitellin, a phosphoprotein, which binds calcium in a non-ionized, physiologically inactive, form. This enables the organism to tolerate a large increase in the level of the serum calcium. In the mouse, during the period of most extensive endosteal-bone formation, there was no increase in the level of the serum calcium or phosphorus. As far as is known, mammals which give birth to their young alive do not possess anything like the serum vitellin, and do not show any appreciable elevation in the level of the serum calcium in response to estrogen. It is also of particular interest that the serum alkaline phosphatase was not changed during the endosteal reaction of the mouse skeleton, as it is in Paget's disease, malignant bone tumors, rickets, and other conditions in which there is increased osteoblastic activity.

Estrogens have been found to have local action in producing the morphological changes of estrus in isolated slices of the uterine mucosa. In the experiments described, implants of the free-steroid hormones had no local effects upon bone tissue. Pfeiffer studied the effect of estrogen upon ectopic bone formation in marrow transplants in the testes, but could find no production of endosteal new bone in mice.

SUMMARY AND CONCLUSIONS

1. Endosteal new-bone formation was studied in estrogen-treated mice, including observations upon the gross, microscopic, and roentgenographic features of the skeletal reaction.

2. A quantitative study by means of combined roentgenographic and histological techniques gives accurate and uniform results in the measurement of the osteogenetic reaction of the skeleton.

3. The pattern, sequence, and rate of the bone changes in estrogen-treated mice are similar to those of the growth-rate differential of the skeleton.

4. Newborn mice, treated with estrogen from the date of birth, show microscopic evidence of changes within ten days, although no gross or roentgenographic changes are demonstrable until after twenty days of treatment. The skeletons of embryos of treated mothers show no endosteal bone in roentgenograms.

5. Mice from one to six months of age show no appreciable differences in their response to estrogens. Aged mice, one year of age or older, show some delay in the response, and the volume of endosteal bone is less than in similarly treated young mice. There was no variation in the bone reaction in the different hereditary strains studied.

6. The volume of new bone deposited is, within the limits of the tolerance of the animal, directly proportional to the dose and the period of administration.

7. Mice, treated with 2.0 milligrams of α-estradiol benzoate over a period of four weeks, recovered from the effects of estrogen after treatment was discontinued. Normal endochondral ossification reappeared below the epiphyseal lines within six weeks. The endosteal bone was almost completely absorbed within eighteen weeks.

8. The serum calcium, inorganic phosphorus, and alkaline phosphatase are within the range of normal levels during administration of estrogen and endosteal new-bone formation.

9. Esterification of an estrogen is one of the most important factors in its osteogenetic potency.

10. Endosteal new-bone formation is not induced by lutoid, corticoid, or testoid hormones. There is neither inhibitory nor synergistic action of progesterone, desoxycorticosterone acetate, or testosterone propionate upon the osteogenetic action of estrogens.

11. Osteogenetic action of estrogen is neither enhanced nor inhibited by simultaneous treatment of the animals with preparations of anterior-pituitary hormone. Adrenocortitotropic hormone induces no endosteal new-bone formation.

12. No evidence has been found that the mouse produces endosteal new bone, characteristic of the effects of estrogens, under strictly physiological conditions. During pregnancy, when large amounts of estrogen are being excreted in the urine, neither the foetal nor the maternal skeleton shows roentgenographic evidence of estrogen-induced bone formation. No production of endosteal bone has been observed in either immature or mature mice in which the ovaries have been under the influence of large doses of gonadotropic hormones, administered over a long period of time.

13. No specific local osteogenetic effects from implantation of free estrogens in bones have been observed. The skeleton showed the usual systemic osteogenetic reaction to absorption of implants of estrogen, but not to the control implants of cholesterol.

14. Evidence has been submitted which suggests that the estrogen-induced bone changes are a reaction to injury of the osteogenic connective tissues of the primitive bone marrow.

15. These findings constitute a demonstration of experimental marble-bone disease. The experimental condition in mice is like the disorder observed by Albers-Schönberg in the following ways: (1) The entire skeleton shows unabsorbed cartilage matrix in the bone tissue of the growing metaphyses; (2) the serum alkaline phosphatase, calcium, and inorganic phosphorus are within the normal range; (3) there is no appreciable osteoclast formation or bone resorption; (4) withdrawal of the causative factor reinstates the normal balance between bone formation and bone resorption, and results in a roentgenographic picture similar to marble bones during a period of remission of the disease.

REFERENCES

1. Bloom, William; Bloom, M. A.; and McLean, F. C.: Calcification and Ossification. Medullary Bone Changes in the Reproductive Cycle of Female Pigeons. Anat. Rec., **81**: 443–475, 1941.
2. Bodansky, Aaron: Phosphatase Studies. II. Determination of Serum Phosphatase. Factors Influencing the Accuracy of the Determination. J. Biol. Chem., **101**: 93–104, 1933.
3. Clark, E. P., and Collip, J. B.: A Study of the Tisdall Method for the Determination of Blood Serum Calcium with a Suggested Modification. J. Biol. Chem., **63**: 461–464, 1925.

4. Evans, J. S.; Varney, R. F.; and Koch, F. C.: The Mouse Uterine Weight Method for the Assay of Estrogens. Endocrinology, **28:** 747–752, 1941.
5. Fairbank, H. A. T.: Osteopetrosis. Osteopetrosis generalisata, Marble bones, Albers-Schönberg's disease, Osteosclerosis fragilitas generalisata. J. Bone and Joint Surg., **30-B:** 339–356, May 1948.
6. Gardner, W. U.: Sexual Dimorphism of the Pelvis of the Mouse. The Effect of Estrogenic Hormones upon the Pelvis and upon the Development of Scrotal Hernias. Am. J. Anat., **59:** 459–483, 1936.
7. Gardner, W. U.: Influence of Sex and Sex Hormones on the Breaking Strength of Bones of Mice. Endocrinology, **32:** 149–160, 1943.
8. Gardner, W. U.: Effect of Steroids. Conference on Metabolic Aspects of Convalescence Including Bone and Wound Healing, **4:** 21–22, 1943.
9. Gardner, W. U.: Further Studies on the Effects of Estrogen on Bone Formation in Mice. Trans. Conference on Metabolic Aspects of Convalescence, **12:** 79–97, 1946.
10. Gardner, W. U., and Clouet, D. H.: Osseous Proliferation in Estrogen-Treated Hypophysectomized Mice. (Abstract.) Anat. Rec., **88:** 433, 1944.
11. Gardner, W. U., and Clouet, D. H.: Influences of Estrogens upon the Bones of Young and Old Mice and of Thyroid Deficient Mice. (Abstract.) Anat. Rec., **91:** 275, 1945.
12. Gardner, W. U., and Pfeiffer, C. A.: Inhibition of Estrogenic Effects on the Skeleton by Testosterone Injections. Proc. Soc. Exper. Biol. and Med., **38:** 599–602, 1938.
13. Gardner, W. U., and Pfeiffer, C. A.: Influence of Estrogens and Androgens on the Skeletal System. Physiol. Rev., **23:** 139–165, 1943.
14. Gomori, G.: A Modification of the Colorimetric Phosphorus Determination for Use with the Photoelectric Colorimeter. J. Lab. and Clin. Med., **27:** 955–960, 1942.
15. Jacobson, L. O.: The Effect of Estrogens on the Peripheral Blood and Bone Marrow of Mice. Endocrinology, **34:** 240–244, 1944.
16. Krohn, P. L.: The Prolonged Administration of Oestrogen or Androgen to Immature Female Monkeys. J. Endocrinol. (Proc. Soc. Endocrinology), **5:** xci–xcii, 1948.
17. Kyes, Preston, and Potter, T. S.: Physiological Marrow Ossification in Female Pigeons. Anat. Rec., **69:** 377–379, 1934.
18. McLean, F. C., and Bloom, W.: Calcification and Ossification. Mobilization of Bone Salt by Parathyroid Extract. Arch. Pathol., **32:** 315–333, 1941.
19. Miller, E. W.; Orr, J. W.; and Pybus, F. C.: The Effect of Oestrone on the Mouse Skeleton, with Particular Reference to the Newcastle Bone Tumour (NBT) Strain. J. Path. and Bact., **55:** 137–150, 1943.
20. Pfeiffer, C. A.: Development of Bone from Transplanted Marrow. Conference on Metabolic Aspects of Convalescence Including Bone and Wound Healing, **6:** 88–92, 1944.
21. Reed, C. E.; Reed, B. P.; and Gardner, W. U.: The Influence of Estrogens on the Crystal Structure of Bone as Revealed by X-Ray Diffraction Studies on the Femora of Mice. Endocrinology, **38:** 238–244, 1946.
22. Segaloff, Albert, and Cahill, W. M.: Endosteal Bone Deposition in Femurs of Vitamin D-Deficient Mice Treated with Estrogen. Proc. Soc. Exper. Biol. and Med., **54:** 162–163, 1943.
23. Silberberg, Martin, and Silberberg, Ruth: Combined Effects of an Estrogen and an Anterior Hypophyseal Extract on the Skeleton of the Growing Mouse. Arch. Pathol., **39:** 381–387, 1945.
24. Silberberg, Martin, and Silberberg, Ruth: The Influence of Sex and Breeding on Skeletal Ageing of Mice. Anat. Rec., **91:** 89–101, 1945.
25. Silberberg, Martin, and Silberberg, Ruth: Skeletal Changes Caused by the Combined Administration of Thyroxin and Estrogen. Am. J. Pathol., **22:** 1033–1045, 1946.
26. Sotiriadou, E.: Effect of Oestrogens on Hypophysectomized Female Mice. Acta Brevia Neerlandica, **10:** 177–179, 1940.
27. Sutro, C. J., and Pomerantz, Leo: Effects of Estrogen on Bones, Joints and Ligaments of Castrated Guinea Pigs. Arch. Surg., **39:** 992–1000, 1939.
28. Urist, M. R.; Bell, R., Jr.; and Voshell, A. F.: Unpublished Studies on Two Cases of Osteogenesis Imperfecta Treated with Large Doses of Estrogens.
29. Urist, M. R.; Budy, A. M.; and McLean, F. C.: Factors Influencing the Reaction of the Mammalian Skeleton to Estrogens. Trans. Conference on Metabolic Aspects of Convalescence, **17:** 79–105, 1948.
30. Urist, M. R.; Budy, A. M.; and McLean, F. C.: Species Differences in the Reaction of the Mammalian Skeleton to Estrogens. Proc. Soc. Exper. Biol. and Med., **68:** 324–326, 1948.
31. Urist, M. R., and McLean, F. C.: Calcification and Ossification. I. Calcification of the Callus in Healing Fractures in Normal Rats. J. Bone and Joint Surg., **23:** 1–16, Jan. 1941.
32. Wentworth, J. H.; Smith, P. K.; and Gardner, W. U.: The Composition of Bones of Mice Receiving Estrogens and Androgens. Endocrinology, **26:** 61–67, 1940.

AN END-RESULT STUDY OF THE KELLER OPERATION *

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In 1927, Cleveland reported a series of 200 operations for the relief of hallux valgus and hypertrophic arthritis of the metatarsophalangeal joint. This earlier follow-up study, at The New York Orthopaedic Dispensary and Hospital, was undertaken with relatively limited personal experience, so that certain conclusions which were reached need revision or correction. Changes in surgical techniques are bound to occur as experience accumulates; there should be no reluctance to admit a change of ideas with the passage of time.

Various surgical attempts to relieve hallux valgus or hallux rigidus have been proposed, some of which are sound and others of questionable value. The general practice of those who were trained or worked at The New York Orthopaedic Hospital during the decade from 1921 to 1930 was, for the most part, simply to excise the exostosis on the first metatarsal head and the adjacent lip of the proximal phalanx. Although this procedure relieved the pain of the inflamed bursa, the underlying hallux valgus was not improved. For a stiffened arthritic joint, the operation is useless.

Dudley Morton[4], in his investigation of the causes of foot pain, lists the short first metatarsal, sesamoids placed posteriorly beneath the head of the first metatarsal, and relaxation of the joint between the first and the second cuneiform bones as structural factors causing pain in the foot. He established the weight-bearing ratio of the metatarsal heads as 2–1–1–1–1 from the first to the fifth metatarsal [5] and, with this fact, the concept of an anterior transverse arch has disappeared.

Any operative procedure which radically upsets this weight-bearing ratio should be expected to produce more discomfort than alleviation of symptoms. An excision of the first metatarsal head or its underlying sesamoid bones should be avoided, except for urgent necessity.

About fourteen years ago we began, at St. Luke's Hospital, to utilize the arthroplastic operation described by Keller for correction or relief of hallux valgus and chronic hypertrophic arthritis of the metatarsophalangeal joint of the great toe. It has proved to be an extremely satisfactory surgical approach to the problem. The patient with painful calluses beneath the second and third metatarsal heads, or metatarsalgia, or the infrequent plantar neuroma, should be advised that surgical correction of hallux valgus *per se* will not relieve these symptoms. Excision of the proximal phalanx of a hammer toe will, however, often alleviate this metatarsal pain.

The authors have not found that posterior displacement of the sesamoid bones upsets the weight-bearing ratio sufficiently to be the cause of pain in the fore part of the foot. With the Keller procedure, in every instance the distal insertions of the medial and lateral heads of the flexor hallucis brevis are divided and the sesamoid bones invariably retract to a posterior position (Figs. 1-A and 1-B), yet in only one of the 113 patients studied did serious postoperative metatarsalgia develop.

The surgical correction of symptoms due to hallux valgus or hypertrophic arthritis of the great toe, so-called hallux rigidus, is not to be undertaken lightly. If the patient is an athlete, the operative procedure may cause his retirement from active competitive sports. Among young men of the armed forces, it frequently means a limited-service status or discharge for disability [2].

Sex

In this series and in that presented earlier, 90 per cent. of the patients operated upon were women. The authors believe that there is no such discrepancy between the sexes as

* Read at the Annual Meeting of The American Orthopaedic Association, Colorado Springs, Colorado, May 21, 1949.

TABLE I

OPERATIONS ON 113 PATIENTS *

Operation	Hallux Valgus		Hallux Rigidus	
	No. of Patients	No. of Operations	No. of Patients	No. of Operations
Unilateral	17	17	20	20
Bilateral	73	146	5	10
Total	90	163	25	30

* Two patients in this series were included in both groups. Each had both conditions, in opposite feet.

to actual incidence of these disabilities. Men are probably willing to accept broader and more sensible shoes to relieve their discomfort.

Age

The youngest patient was fourteen and the oldest eighty-two, with an average for the entire group of forty-eight years. This in itself means nothing, except to indicate at what age the patients decided to seek surgical relief. Hallux valgus was doubtless noted in early life; hallux rigidus, a wear-and-tear form of arthritis, begins to cause trouble in middle life.

FOLLOW-UP STUDY

Approximately 312 Keller operations have been performed by members of the Orthopaedic Staff at St. Luke's Hospital. All cases which have been followed for less than one year have been discarded from this study. The patients included have been personally examined and the results checked by roentgenograms. This report is based on the records

FIG. 1-A FIG. 1-B

Typical preoperative and postoperative views of hallux valgus. A Keller arthroplasty was performed. There is slight narrowing of the foot, due to removal of the exostosis, and slight approximation of the first metatarsal toward the second metatarsal. The correction of the hallux valgus is due to removal of the adductor attachment of the proximal phalanx. The sesamoid bones retracted after operation.

of 113 patients upon whom 193 operations were performed (Table I); their postoperative course was followed for an average of twenty-three months after surgery, during the period from 1938 to 1948, inclusive. Ninety of these patients had hallux valgus and twenty-five had hallux rigidus, a ratio of almost four to one. Of the patients with hallux valgus, seventy-three, or 81 per cent., had bilateral deformity. Hallux rigidus is much more apt to be confined to a single foot. In only five of the twenty-five patients was this disability bilateral,—an incidence of 20 per cent.

CARE OF THE PATIENTS

Operative Care

No attempt will be made to describe in detail the operative technique, but certain points should be stressed in the operative and postoperative care. In the vast majority of patients, the operation is performed with a tourniquet. In the presence of varicose veins, or where there is a history of phlebitis, it may be advisable to omit the tourniquet. Soft tissue should be separated from underlying bone by sharp dissection. A subperiosteal removal of bone, such as Keller originally recommended in 1904, invites regeneration of the excised phalanx or exostosis. At least one-half of the phalanx should be excised to prevent the postoperative development of hallux rigidus. All chips of bone should be carefully removed from the wound and all cut bone edges should be smoothed.

Postoperative Care

A rather long period of convalescence is required. We recommend that these patients remain in the hospital from fifteen to eighteen days, or that they are non-ambulatory for a period of at least fourteen days. The patient should be instructed to begin moving the toes twenty-four hours after operation. Wound healing is slow. The sutures should not be removed until the twelfth to the fourteenth postoperative day, and weight-bearing should not be permitted until the fifteenth postoperative day. The patients are unable to tolerate a regular shoe for about one month. A shoe split on the medial side, along the margin of the sole, provides more room for the swollen, tender joint. Metatarsal bars, if properly placed and of sufficient height, are a benefit in lessening pain due to weight-bearing. Those patents with sedentary occupations were able to return to work between the third and fourth week. Patients who, by necessity, had to be on their feet for long periods of time, or whose jobs required considerable walking, were unable to carry out their duties until six to eight weeks had elapsed. Many patients who followed a normal postoperative course experienced discomfort for as long as six months after surgery, when they indulged in unlimited activity.

END RESULTS

The end results were considered from the point of view of the surgeon and from the opinion expressed by the patient. The surgeons' evaluation of the 193 operations was as follows:

	No.	*Per cent.*
Excellent	23	12
Good	156	81
Poor	14	7

This is an arthroplastic operation, and return to normal function should not be expected in most instances. An *excellent* result is one in which there is a normal range of active motion in the great toe, with correction of valgus or elimination of hallux rigidus. This normal range of active motion is seldom obtained. A *good* result is one in which there is a fair range of active motion,—that is, dorsiflexion of 30 degrees, plantar flexion of 10

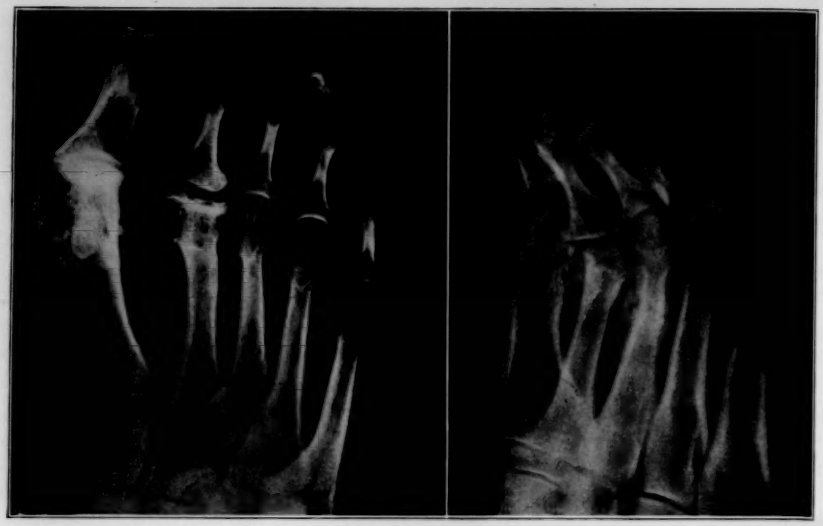

FIG. 2-A FIG. 2-B

Fig. 2-A: Preoperative view of hallux rigidus with infraction of the second metatarsal head.
Fig. 2-B: Postoperative oblique view of the same foot, showing march fracture which occurred six months after surgery.

degrees, and less than 10 degrees of hallux valgus. In addition, there is ample passive motion, so that the great toe is unrestricted in walking. This type of result was achieved in the vast majority of our patients. A *poor* result is one in which the toe shows limited motion,—that is, less than 30 degrees of dorsiflexion and less than 10 degrees of plantar flexion with discomfort due to the restricted motion.

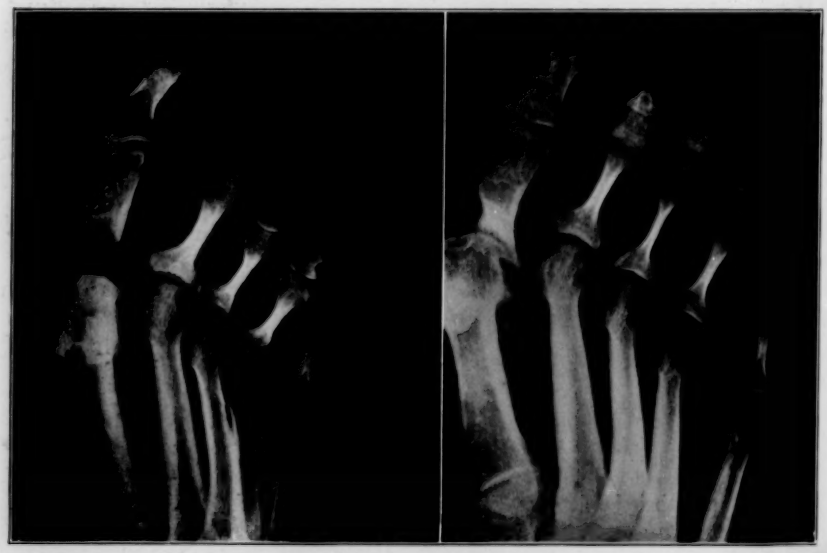

FIG. 3-A FIG. 3-B

Postoperative roentgenograms, taken immediately after surgery and three years later, show regeneration of the proximal end of the resected phalanx.

TABLE II

COMPLICATIONS IN 193 OPERATIONS

Complication	No. of Patients
Delayed wound healing	22
Postoperative infection	6
Persistent pain in great toe	4
Persistent swelling in great toe for 13 months	1
March fracture	2
Pulmonary embolus	1
Pneumonia, Type III	1
Pleurisy, postoperative	1
Acute cholecystitis	1
Acute gout	1

The patients' opinions of their end results were as follows:

	No.	*Per cent.*
Pleased	97	86
Satisfied	11	10
Dissatisfied	5	4

From this it can be seen that the patients, almost unanimously, were pleased with the results of their operations. This is understandable when it is realized that the patient is interested chiefly in freedom from pain. The results in the five patients who were dissatisfied are considered as failures; they account for seven of the fourteen poor operative results, as recorded by the surgeons. The remaining seven poor operative results will be discussed in a consideration of the complications.

Twelve of these patients had had previous surgery for their hallux valgus. Eleven had simple exostectomy of the deformed first metatarsal head; the twelfth patient had resection of the metatarsal head.

FIG. 4-A FIG. 4-B

Fig. 4-A: Preoperative roentgenogram shows cystic degeneration of the head of the first metatarsal.

Fig. 4-B: Roentgenogram taken one year after surgery for hallux rigidus.

In addition to the Keller operation, the following foot surgery was performed simultaneously: Ten patients had excision of the proximal phalanx of the second digit for correction of hammer-toe and alleviation of metatarsalgia. Five patients had exostectomy of the head of the fifth metatarsal, and one had excision of a plantar neuroma between the third and fourth metatarsal heads. Hallux valgus is frequently accompanied by other painful foot conditions, and one should not expect an operation on the great-toe joint to remedy coexisting foot complaints.

COMPLICATIONS

There were no deaths in this series, but pulmonary embolus occurred once.

Delayed Wound Healing and Postoperative Infections

FIG. 5-A

The complication most commonly encountered was delayed wound healing (Table II). Until the end of 1945, sutures were removed on the seventh to the eleventh day after operation. One patient had the sutures removed on the sixth postoperative day; the wound avulsed as soon as weight-bearing was begun on the following day. We were slow to learn that sutures in these wounds should remain undisturbed for twelve to fourteen days, and that no weight-bearing should be allowed for two weeks. In twenty-two operations there was delayed healing of the operative wound. In sixteen of the cases

Fig. 5-A: Hallux valgus, before operation.

Fig. 5-B: Roentgenogram taken immediately after operation.

Fig. 5-C: Roentgenogram, taken one year later, shows some absorption of the remaining portion of the proximal phalanx.

FIG. 5-B FIG. 5-C

maceration of the wound margins occurred without infection or avulsion of the wound. The hospital stay was not prolonged in these cases, if we consider eighteen days the required period of hospitalization for this operation.

The remaining six patients with delayed wound healing all had postoperative infection. In one case, the infection was present at the time of suture removal; in the other five, infection developed in wounds that had avulsed following early removal of sutures and weight-bearing. These patients required prolonged periods of hospitalization, varying from twenty-one to eighty-five days. One of the six had diabetes of long standing; he had been treated for two months prior to his operation in an effort to clear an infected bursa overlying a hallux rigidus. The sutures were removed on the ninth postoperative day, and the wound broke down with loss of a large area of skin. The infection was controlled and, four weeks following operation, twenty-four pinch grafts were required to cover the denuded area. The wound finally healed and the patient was discharged on the sixty-fourth postoperative day. This was the only patient with diabetes, of the five operated upon, whose wound became infected; he has had a poor result with very limited motion, but no pain.

All six patients with postoperative infection had stiffness of the great-toe joint; in five the results were considered poor by the surgeon and, as previously mentioned, one was a failure. The average period of hospitalization for these patients with infected wounds was almost fifty days.

March Fractures

March fractures or insufficiency fractures were noted twice (Table II). Both patients complained of severe metatarsalgia,—one at two and one-half months, and the other six months following surgery. There was no history of injury in either case. The fractures were not suspected, but roentgenograms were taken in each instance because of the severity of the patient's complaints. One had a fracture of the second metatarsal shaft, while the other had a fracture in the shaft of the third metatarsal. This patient, in addition to the hallux valgus, had infraction or osteochondritis of the second metatarsal head, treated surgically (Figs. 2-A and 2-B).

FIG. 6-A

Preoperative rentgenograms show hallux valgus with short first metatarsals.

POSTOPERATIVE BONE CHANGES NOTED BY ROENTGENOGRAM

Every patient had preoperative as well as postoperative roentgenograms. It would be discouraging to evaluate the end result of this procedure entirely on the basis of a roentgenographic study. Many patients with unfavorable roentgenographic changes had no symptoms.

If resection of a portion of the phalanx is done subperiosteally, regeneration of the proximal end of the phalanx may occur (Figs. 3-A and 3-B). This was noted in four post-

FIG. 6-B

Roentgenograms, taken immediately after operation, show that a fragment of bone has been left behind, medial to the head of the right metatarsal. Otherwise the condition appears satisfactory.

FIG. 6-C

One and one-half years after operation, roentgenograms show cystic degeneration of the head of the first metatarsal bilaterally.

FIG. 6-D

Fig. 6-D: Roentgenograms, taken six and one-half years after the Keller arthroplasty and five years after attempt to arthrodese the metatarsophalangeal joints, show fusion on the left and pseudarthrosis on the right. The wire for fusion of the first metatarsophalangeal joint is still *in situ*. The left foot was asymptomatic; the right remained painful.

Fig. 6-E: Final roentgenogram of the right foot shows that the wire has been removed; pseudarthrosis persists.

FIG. 6-E

operative roentgenograms. Fortunately, in these cases no symptoms developed, but if the original condition had been hallux rigidus, the condition would have tended to recur.

Cystic degeneration of the head of the first metatarsal was encountered in five instances (Figs. 4-A and 4-B). Four of these patients had hallux rigidus, and these changes are probably related to the existing arthritis.

There were two instances in which partial absorption of the remaining portion of the proximal phalanx occurred after operation (Figs. 5-A, 5-B, and 5-C). In neither case did the patient have symptoms resulting from the absorption.

FAILURES

Operative failure is more prone to occur in hallux rigidus, when an inadequate resection of the proximal phalanx has been performed. The results in five patients, upon whom six operative procedures were carried out, must be considered as failures, because of persistent pain, either alone or in combination with stiffness of the joint which had been operated upon.

CASE 1. L. G., a woman, forty-five years old, is still having pain in the right great toe and fore part of the foot, seven years after her original surgery. This patient had a bilateral Keller operation in January 1942,

FIG. 7-A

Preoperative roentgenograms show moderately severe bilateral hallux valgus.

FIG. 7-B

Roentgenograms, taken one year after operation, show only partial correction of the hallux valgus on the left. The hypertrophic sesamoid bone may be seen, underlying the first metatarsal head in each foot.

for hallux valgus (Figs. 6-A and 6-B). The wounds healed well and she was discharged on the seventeenth day after operation. Her postoperative roentgenograms appeared satisfactory. She was given exercises for her toes, and at a later date metatarsal bars were applied to her shoes. In June 1943, one and one-half years after the operation, the patient was still having considerable pain in both great toes. There was limited motion in the great toes, and roentgenograms showed cystic degeneration of the first metatarsal head bilaterally (Fig. 6-C). Since the short first metatarsal is a cause of metatarsalgia, fusion of the metatarsophalangeal joint of the great toe was done in an attempt to lengthen the first metatarsals.

Five years later the patient returned to the Out-Patient Department, complaining of metatarsalgia and pain in the scar of the Keller incision on the right. In June 1948, six and one-half years following the Keller arthroplasty and five years after the attempt to arthrodese the metatarsophalangeal joints (Fig. 6-D),

the wire was removed from the right great toe and a neuroma in the scar was excised. Pseudarthrosis of the metatarsophalangeal joint was present (Fig. 6-E). The left great toe was fused solidly and did not produce symptoms. When last seen, nearly seven years following the original Keller operation, the patient still had pain in her right great toe and fore part of the foot. Motion was still present in her right first metatarsophalangeal joint. This rather unusual operation was successful in lengthening the first metatarsal and eliminating metatarsalgia on one side.

CASE 2. The next failure occurred in I.S., a woman, sixty-two years old. A bilateral Keller operation was performed in June 1942, for moderately severe hallux valgus (Fig. 7-A). Twenty-nine months after operation

FIG. 8-A

Preoperative roentgenograms show bilateral hallux rigidus.

FIG. 8-B

Roentgenograms, taken six months after the Keller arthroplasty, show insufficient removal of the proximal phalanx.

FIG. 8-C

The final roentgenograms, taken five and one-half years after operation, show ankylosis of the first metatarsophalangeal joint on the right and recurrence of the hallux rigidus on the left.

the patient was still having pain, and all supportive measures had failed to alleviate her complaints. There was only partial correction of the hallux valgus on the left, and the great toe was held in 10 degrees of dorsiflexion. In addition to the persistent hallux valgus, the sesamoid bone underlying the first metatarsal head was hypertrophic (Fig. 7-B).

CASE 3. A failure occurred in a woman, sixty-six years old, who had complained of pain in both feet for many years. A Keller arthroplasty was performed on the left foot in August 1943. The wounds healed by first intention, and the patient was discharged twenty-one days after operation. She continued to have pain; twenty-seven months after the operation, she still complained of pain in association with weight-bearing. Her last roentgenograms, two years after operation, showed nothing remarkable, although there was evidence of cystic changes in the heads of the first metatarsals. Shortly after her last visit to the Orthopaedic Clinic, this patient was admitted to the hospital for an emergency abdominal operation; she died of uraemia.

CASE 4. A man, forty-seven years old, complained of pain three years after he had had a Keller arthroplasty for hallux rigidus. Examination showed that some rigidus had recurred in his metatarsophalangeal joint. This patient had returned, one and one-half years after the operation, complaining of pain; and he reported at that time that he had dropped a cooking pot on his great toe.

CASE 5. The fifth failure occurred in a woman, fifty-one years old, who had bilateral hallux rigidus (Fig. 8-A). Postoperatively the wound on the left failed to heal promptly and there was a two-millimeter area of sloughing in the center of the incision, which required thirty-one days to heal. In three months the patient was walking approximately one-half mile, and had little pain beneath the first metatarsal head on the left.

Six months after her operation she had an excellent range of motion and no pain associated with walking. She was very pleased. However, the postoperative roentgenograms (Fig. 8-B) show that an insufficient amount of the proximal phalanx had been resected bilaterally.

When next seen, five and one-half years later, the patient stated that her toes were fine, but that she had pain beneath the second and third metatarsal heads bilaterally and a painful callus beneath the right great toe. When examined, she had no motion in the metatarsophalangeal joint of the right great toe and practically none in the left. The final roentgenograms show ankylosis of the first metatarsophalangeal joint on the right with recurrent hallux rigidus on the left (Fig. 8-C). In spite of the patient's opinion that her toes were fine, the result must be considered a failure.

CONCLUSIONS

1. Arthroplasty of the metatarsophalangeal joint of the great toe by the Keller procedure gave good or excellent results in 93 per cent. of 193 operations in this series.

2. Only five of the 113 patients were dissatisfied, or had complete operative failures.

3. The chief complication has been delayed wound healing, caused by early removal of sutures and by weight-bearing before the fourteenth day after operation.

4. A long period of convalescence is required after this procedure.

REFERENCES

1. Cleveland, Mather: Hallux Valgus. Final Results in Two Hundred Operations. Arch. Surg., **14**: 1125–1135, 1927.
2. Cleveland, Mather; Willien, L. J.; and Doran, P. C.: Surgical Treatment of Hallux Valgus in Troops in Training at Fort Jackson During the Year of 1942. J. Bone and Joint Surg., **26**: 531–534, July 1944.
3. Keller, W. L.: The Surgical Treatment of Bunions and Hallux Valgus. New York Med. J., **80**: 741–742, 1904.
4. Morton, D. J.: Structural Factors in Static Disorders of the Foot. Am. J. Surg., **9**: 315–326, 1930.
5. Morton, D. J.: Foot Disorders in General Practice. J. Am. Med. Assn., **109**: 1112–1119, 1937.

CHRONIC NON-SPECIFIC TENOSYNOVITIS WITH EFFUSION ABOUT THE ANKLE

Report of Three Cases

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The non-specific inflammatory lesions of the tendons, to which this discussion will be limited, may be divided into three groups: (1) peritendinitis crepitans; (2) stenosing tenosynovitis; and (3) chronic tenosynovitis with effusion.

Peritendinitis Crepitans

In peritendinitis crepitans, the deposition of fibrin in the peritendinous and also the perimuscular areolar tissue produces characteristic crepitation on motion. The condition is usually of an occupational nature, and is caused by frequent, prolonged repetition of the same motion. It is particularly prone to develop in a person who is unaccustomed to a new type of work, or in an old worker who is out of training[13]. The crepitus is more marked in the morning and disappears after use of the limb. The condition may also follow trauma, infection, or a combination of both.

Cohen and Reid reported seven cases of peritendinitis crepitans associated with oxaluria, in all of which the tenosynovitis cleared a few weeks after institution of a diet to control the oxaluria, combined with physical therapy.

Howard performed biopsy in three of his thirty-two cases of peritendinitis crepitans; he found acute degenerative muscle changes, but the tendon sheaths were uninvolved. He emphasized: "The disease is not connected with the synovial tendon sheaths, and is not a 'synovitis sicca'". Rest and physical therapy invariably relieve the condition within a few days.

Stenosing Tenosynovitis

Stenosing tenosynovitis, of course, occurs only in tendons possessing a synovial sheath, with the constriction located at the narrowest portion of that sheath. One of the common locations for stenosing tenosynovitis is at the radial styloid, where both the abductor pollicis longus and the extensor pollicis brevis share the same sheath. The important action of these two muscles as stabilizers of the thumb, together with the narrowness of the channel through which both of their tendons pass, lying directly on the bone and being surrounded by tough, unyielding fibers of the annular ligament, create favorable conditions for stenosis of this sheath[5].

A similar anatomical and mechanical situation, producing "trigger-finger", exists on the palmar aspect of the metacarpals, just proximal to the metacarpophalangeal joint, where the tendons of the flexor digitorum sublimis and the flexor digitorum profundus pass through a narrow sheath, lying close to the bone and bound by the unyielding crucial and transverse fibers of the palmar fascia.

Frequently repeated action of the same muscles produces inflammatory changes, with subsequent fibrosis and hourglass constriction of the tendon sheath and of the tendon[9].

Although in early cases (less than four weeks) immobilization for three or four weeks may occasionally relieve the symptoms, surgery is usually required in the older cases. The operation is comparatively simple and is usually performed with local anaesthesia, the patient leaving the hospital immediately thereafter and returning to work two or three weeks later. In a small number of the authors' cases, the symptoms subsided spontaneously in a few months.

Hackenbroch, in 1927, reported two cases of typical post-traumatic stenosing tenosynovitis with an unusual location in the peroneal sheath, below the lateral malleolus. Both were relieved by the operation. Jagerink, in 1928, also obtained complete cure after operative release of a stenosing tenosynovitis of the anterior tibial sheath, which is a most unusual location.

Chronic Tenosynovitis with Effusion

The third group, tenosynovitis with effusion, is the one to which all of the cases reported here belong. Most commonly, chronic effusion in the synovial sheath is due to tuberculosis, and is observed on the dorsum of the hand or foot or in the palmar synovial sheaths of the wrist. The synovial sheath is distended with fluid and contains typical rice bodies. Occasionally, single or multiple foci of tenosynovitis may precede or be associated with rheumatoid polyarthritis; this has been observed in cases with Still's syndrome[2]. Tuberculosis and suppurative forms of tenosynovitis are not within the scope of this paper.

Non-specific chronic tenosynovitis with effusion must be considered a rarity, particularly at the ankle. A thorough search of the literature* disclosed only one case somewhat similar to the authors' Cases 2 and 3, that reported by Kulowski, in which the posterior tibial tendon sheath was affected. No case was found of tenosynovitis of the flexor hallucis longus, resembling Case 1.

Volk reported sixty-three cases of "traumatic tenosynovitis", found in Army training camps. All of his patients returned to full duty after an average of five days of rest and

* Quarterly Cumulative Index Medicus, Volumes 1 to 41, inclusive (January 1927 through June 1947), were checked under the headings of "Foot" and "Tendon".

physical therapy. The Achilles tendon was most commonly affected,—in forty-two cases. Other tendons of the foot included the extensor hallucis longus in nine cases; the tibialis anterior in five cases; the tibialis posterior in one case; and the peronaeus in one case. All of Volk's cases belonged to our first group,—namely, peritendinitis crepitans.

Lipscomb studied 190 cases of non-specific tenosynovitis, mainly of the hand and foot. In only fifteen cases were the patients operated upon and pathological studies made. Lipscomb stated: "In one case definitely murky fluid was present under pressure". Of the entire series, twelve cases were recorded in which the posterior tibial tendon was involved, and three cases in which the flexor hallucis longus tendon was involved; the series included cases of peritendinitis crepitans, stenosing tenosynovitis, and one case which was apparently tenosynovitis with effusion. On microscopic study, definite pathological changes were found in the tendon sheaths, with various stages of chronic inflammation. Unfortunately, Lipscomb does not specify which of his cases were treated surgically.

Bonnet presented one case in which he postulated that pressure by an unusually large trochlear process of the calcaneus produced what he thought to be peroneal tenosynovitis. The condition was relieved by Whitman plates. Pollosson, in discussing Bonnet's presentation, recalled two similar cases, in one of which the enlarged trochlear process had been removed, but the peroneal synovial sheath had appeared to be normal.

Aberle-Horstenegg called attention to a painful condition over the sole of the foot, due to tenosynovitis of the distal synovial sheath of the peronaeus longus tendon, near its insertion into the base of the first metatarsal.

In view of the apparent infrequency of non-specific tenosynovitis at the ankle, the authors wish to record the findings in three patients, the only ones known to have been operated upon for this condition at the Hospital for Joint Diseases.

CASE 1. H. F., a physician, thirty-five years old, was first seen in August 1946. Examination showed a slightly tender, diffuse swelling behind the left medial malleolus. Motions of the foot were free and there was no limp. About one and one-half years before, slight lameness had developed, and pronounced swelling and pitting on pressure had appeared around the right ankle, particularly over its medial aspect. There was no preceding injury or illness. The condition had gradually subsided two or three months later. In the summer of 1946, severe pain developed around the left medial malleolus, and the patient was compelled to discontinue athletics.

The past history was not contributory, except that at the age of fifteen years, hemoptysis had suddenly developed, lasting for twenty-four hours. The patient made a complete recovery after being in bed for six weeks, and had never had recurrence of the hemoptysis. Subsequent clinical study had excluded the possibility of tuberculosis. The patient had always enjoyed good health, and was quite active in athletics.

Roentgenograms of the left ankle and laboratory studies, including the blood Wassermann reaction and sedimentation rate, were normal. Strapping of the ankle failed to relieve the symptoms. Roentgenograms taken in October and November showed "slight calcification" in the soft tissue over the medial aspect of the ankle. Deep roentgenotherapy and local infiltrations with novocain solution produced no effect. Attempted aspiration over the medial aspect of the ankle by another orthopaedic surgeon failed to produce any fluid.

When examined on January 18, 1947, the patient appeared in excellent general health. His gait was normal and he was able to walk on his toes, heels, and the lateral borders of his feet without difficulty. He could squat well, and had no discomfort when hopping on the toes of either foot. The feet were well formed, and there was good pulsation of the arteries. There was one-quarter inch of atrophy of the left calf and a half-inch enlargement of the left ankle. Moderately tender fullness and slightly increased local heat were noted behind the left medial malleolus; the tenderness seemed to correspond with the location of the synovial sheath of the flexor hallucis longus.

Forceful active plantar flexion of the left big toe against passive resistance constantly produced pain over the same area. Since similar testing of the posterior tibial muscles and the flexor digitorum longus produced no pain, and since all other motions of the foot, both active and passive, were free and painless, a tentative diagnosis was made of "benign tumor or chronic tenosynovitis of the flexor hallucis longus". The possibility of infection with Koch's bacillus or of hemangioma of the sheath was considered.

Additional roentgenograms showed slight soft-tissue enlargement and slight calcification and tortuosity of blood vessels behind the left medial malleolus. Similar calcification of blood vessels was present on the normal right side. Exactly the same changes were found when all previous roentgenograms of the left foot were compared.

On March 28, the patient was operated upon at the Hospital for Joint Diseases, general anaesthesia being used. Without a tourniquet, the synovial sheath of the flexor hallucis longus was exposed through a seven-inch incision, curving posteriorly around the medial malleolus. The synovial sheath of the flexor hallucis longus above the posterior process of the talus was found to be distended to about the thickness of a little finger, and contained about 2 cubic centimeters of yellow fluid. Its wall was thickened and there was purplish discoloration of the tendon and the synovial surface of the sheath. The tendon itself was not enlarged, but had lost its normal pearly appearance. The synovial sheath was excised without damage to the posterior ligaments, the channel for the tendon at the sulcus of the flexor hallucis longus over the talus being undisturbed.

The patient was discharged from the Hospital on the sixth postoperative day, walking with crutches, without any immobilization of the left foot. The pathological diagnosis was "non-specific low-grade tenosynovitis". There was no evidence of tuberculosis.

On April 17 (three weeks after the operation), the patient was able to walk on his toes, and forceful flexion of the left big toe against resistance did not cause pain. He was discharged from observation on May 8, completely free of symptoms, and was allowed gradually to resume athletic activities. At follow-up examination on October 20, 1948 (one year and seven months after the operation), the patient was entirely symptom-free, except that the left foot seemed to tire more quickly than the right after overexertion. This, however, did not prevent him from participating in athletics without discomfort.

Case 2. J. G., a man, fifty-eight years old, consulted Dr. Leo Mayer on April 19, 1944, because of occasional pain over the medial aspect of the left ankle, which had started spontaneously about three years previously. It had been preceded by pain in his shoulders, elbows, and knees, all of which had subsided. The ankle pain was described as "drawing" in character. It was present constantly, but was more pronounced during the first few steps taken after resting; it was relieved by walking. Various forms of treatment, such as physical therapy, injections, and immobilization, gave no relief. The patient's general health had been fair.

Examination revealed that he walked with a slight left-sided limp which increased when walking without shoes, the patient being unable to bear weight on the left tiptoes. Slight swelling, increased local heat, and marked sensitivity were noted around the left medial malleolus, the tenderness appearing to be more marked over the bone. No tenderness was present elsewhere in the ankle. Complete laboratory studies were essentially negative.

Roentgenograms showed an area of absorption over the medial malleolus, measuring one and one-quarter inches in length. The preoperative diagnosis was neoplasm of the left medial malleolus.

On May 10, 1944, the patient was operated upon by Dr. Mayer at the Hospital for Joint Diseases. Exploration of the medial malleolus in the area showing rarefaction revealed no pathological changes. The synovial sheath of the posterior tibial tendon contained a little fluid; most of its synovial surface and a considerable portion of the tendon itself were covered with brownish-red granulations, changes described as strongly suggestive of tuberculosis. The entire sheath was excised. The adjacent posterior surface of the tibia and that of the talus appeared to be softened, and they were removed with a gouge. The ankle joint itself was normal. The ankle was immobilized in a plaster-of-Paris casing.

The postoperative diagnosis was recorded as "tuberculosis of the tibialis posticus sheath and tibia and astragalus". The pathologist's examination (B-32311), however, showed no evidence of tuberculosis in the soft tissue or bone. An inflamed tendon sheath, in some places covered with fibrin, was found. The bone underlying the sheath was eroded in places, and in other places, there was evidence of new-bone formation. The diagnosis was: "Tenosynovitis (rheumatoid ?), ankle region, with erosion and new-bone formation in the neighboring bone".

The patient made a satisfactory postoperative recovery, and his local symptoms were relieved.

On November 16, 1947, at re-examination, Dr. Mayer noted that there had been no return of pain in the region of the operation. Marked sensitivity was found over the talonavicular joint, and there was considerable limitation of subtalar motion. An ankle brace was recommended.

Early in 1948, pain again developed in the various joints, and at the end of May the patient's left knee became painful and swollen. Bloody fluid was aspirated from the knee several times. Roentgenograms on July 7, 1948, showed an area of absorption of the lateral femoral condyle. Exploration was recommended, but the patient failed to return.

Case 3. On March 11, 1948, a Negro houseworker, fifty-two years old, was seen because of pain and swelling of the left ankle. In August 1947 she had noticed a slight swelling on the medial side of the left ankle. The region subsequently became painful, and the pain increased despite various forms of treatment (including physical therapy and injections), so that she had to stop working.

Examination revealed a swelling behind the medial malleolus, which was moderately sensitive to touch. The swelling was not discretely outlined, but was firm. Ankle motion was free in all directions. Roentgenographic examination of the left ankle revealed no evidence of bone changes.

The impression was that the patient had a tumor of the medial aspect of the ankle. At operation on March 23, 1948, an incision, three and one-half inches long, was made on the posteromedial aspect of the

ankle joint, behind the malleolus. The sheath of the tibialis posterior tendon was seen to be bulging. When it was incised, about 1.5 cubic centimeters of clear yellow fluid was evacuated. Exploration of the region showed no tumor. The tendon appeared smooth and normal; the sheath was thickened and part of it was excised. Pathological study showed thickening of the tendon sheath.

The postoperative course was uneventful, and when last seen, in August 1948, the patient stated that she was perfectly well. She was able to work full time and had no pain.

SUMMARY

In chronic non-specific tenosynovitis with effusion, localized at the ankle, the symptoms may be present intermittently and the true nature of the condition may remain unrecognized for months or even years.

In two of the cases reported, the posterior tibial sheath was involved, and in one, the flexor hallucis longus (in back of the ankle). Erosion of the tibia and talus was present in Case 2, probably secondary to inflammation of the posterior tibial sheath. This patient also had pain in various joints; and, about four years after the operation, bloody effusion developed in the knee on the same side. Local symptoms were relieved in all three cases after removal of the synovial tendon sheath.

NOTE: The authors wish to thank Leo Mayer, M.D., for his kind permission to include Case 2, and for his assistance in editing this paper.

REFERENCES

1. ABERLE-HORSTENEGG, WALTHER: Ueber einen eigenartigen Fusschmerz. (Tendovaginitis der distalen Sehnenscheide des Peronaeus longus.) Münchener Med. Wchnschr., **79**: 946–948, 1932.
2. BAUMGARTNER, WALTER: Polytendovaginitis rheumatica. Schweiz. Med. Wchnschr., **76**: 809–811, 1946.
3. BONNET, P.: Synovite des péroniers latéraux en relation avec un tubercule externe du calcanéum exubérant. Lyon Chir., **28**: 476–480, 1931.
4. COHEN, HENRY, and REID, J. B.: Tenosynovitis Crepitans Associated with Oxaluria. Liverpool Med.-Chir. J., **43**: 193–199, 1935.
5. FINKELSTEIN, HARRY: Stenosing Tendovaginitis at the Radial Styloid Process. J. Bone and Joint Surg., **12**: 509–540, July 1930.
6. HACKENBROCH, M.: Eine seltene Lokalisation der stenosierende Tendovaginitis (an der Sehnenscheide bei Peroneen). Münchener Med. Wchnschr., **74**: 932, 1927.
7. HOWARD, N. J.: Peritendinitis Crepitans. A Muscle-Effort Syndrome. J. Bone and Joint Surg., **19**: 447–459, Apr. 1937.
8. HOWARD, N. J.: A New Concept of Tenosynovitis and the Pathology of Physiologic Effort. Am. J. Surg., **42**: 723–730, 1938.
9. HUBER, WILLY: Histologische Befunde bei der Tendovaginitis stenosans. Virchow's Arch. f. Path. Anat., **291**: 745–756, 1933.
10. JAGERINK, TH. A.: Über zwei noch nicht beschriebene Formen von Tendovaginitis stenosans. Ztschr. f. Orthop. Chir., **50**: 703–705, 1929.
11. KULOWSKI, JACOB: Tendovaginitis (Tenosynovitis). General Discussion and Report of One Case Involving the Posterior Tibial Tendon. J. Missouri State Med. Assn., **33**: 135–137, 1936.
12. LIPSCOMB, P. R.: Chronic Nonspecific Tenosynovitis and Peritendinitis. Surg. Clin. North America, **24**: 780–797, 1944.
13. OBOLENSKAJA, A. J., und GOLJANITZKI, J. A.: Die seröse Tendovaginitis in der Klinik und im Experiment. Deutsche Ztschr. f. Chir., **201**: 388–399, 1927.
14. VOLK, F. N.: Traumatic Tenosynovitis in a Training Camp. Milit. Surgeon, **94**: 293–296, 1944.

THE MECHANISM OF ROTATION IN COMBINATION WITH LATERAL DEVIATION IN THE NORMAL SPINE

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In an earlier paper [1], the forces producing rotation in scoliosis were described. The mechanism is twofold. One component is contributed by the shape of the wedged vertebral bodies, which, acting as wedges, are squeezed out laterally toward their convex (higher) sides, causing convex-side rotation. Another component force is contributed by the tendency of muscles and ligaments under tension to describe a straight line. The soft structures about the curved line of spinous processes in a scoliotic spine behave in this manner, deflecting the spinous processes in the middle of the curve toward its concavity, and contributing to the convex-side rotation ** observed clinically.

Of these two component forces, one, the wedge mechanism, may be considered pathological, as it operates only in the wedged spine of structural scoliosis. The other is a mechanism which must operate in all spines, and hence may be considered a physiological mechanism for rotation in the normal spine. It is the purpose of this paper to investigate the combination of rotation with lateral deviation in the *normal* spine, to describe the underlying causes for such rotation, and to discuss some concepts currently accepted.

The most commonly accepted explanation of the combination of rotation with lateral deviation is that of Lovett. He quoted Bigelow, who stated: "The principle of torsion is illustrated by bending a flat blade of grass or a flat, flexible stick in the direction of its width. The center immediately rotates upon its longitudinal axis to bend flatwise in the direction of its thickness. In the same way the spine, laterally flexed, turns upon its vertical axis to yield in its shortest or antero-posterior diameter." (Rogers pointed out that the shortest diameter is not always the anteroposterior diameter throughout the spine.)

Lovett [3] argued that, if a straight flexible rod "is bent in two planes at the same time, torsion must inevitably occur". (Rogers constructed such a rod and found that "a symmetrical flexible rod does not rotate at all, either with side bending in extension or with side bending in flexion".)

Lovett then stated: "The vertebral column is not a straight flexible rod, but one bent in physiological curves in the antero-posterior plane; side bending must therefore inevitably lead to torsion, because it means bending in two planes". This point is illustrated by his familiar rubber-strip analogy. This model yields convex-side rotation in side-bending in flexion, and concave-side rotation in side-bending in extension. A detailed investigation of the principles underlying this behavior will throw light upon the whole problem of rotation.

In Figure 1, *A* represents such a strip. The rubber is not under stress and all edges describe a straight line. If the strip is bent forward into what represents kyphosis (Fig. 1, *B*) the anterior edge is under compression (and is actually compressed in length), and the posterior edge is under tension and is elongated. The least bit of lateral deviation will then lead to the situation shown in Figure 1,*C*. The anterior edge has now almost regained its original length by describing a more acute curve between its two ends (making a wider "detour"); the posterior edge, being under tension, describes a straighter line. This behavior is an example of the fact that a curved line placed under compression (the anterior

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** Concave-side rotation of the spinous processes implies, of course, convex-side rotation of the bodies. The direction of rotation of the bodies is the direction usually considered, and this terminology will be followed in this paper. Rotation, in the sense in which it is used here, is considered to mean rotation of the middle of the curve against both ends.

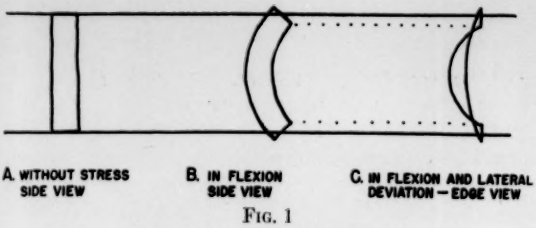

FIG. 1

Behavior of a straight rubber strip.

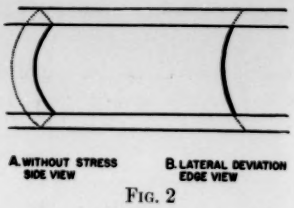

FIG. 2

Behavior of a curved rubber strip.

edge) will become more curved; a curved line subjected to extension (the posterior edge) will become straighter. The difference in the curvature of the two edges results in convex-side rotation. (It will prove advantageous, in this discussion, to keep in mind the concept that rotation is the result of the difference in curvatures between the anterior and posterior "edges" of the spinal column.)

A similar experiment, performed in hyperextension or backward bending of the rubber strip, will lead to concave-side rotation.

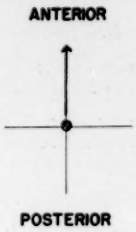

FIG. 3-A

FIG. 3-B

Behavior of a round rod, viewed from above.

If next a strip of rubber is cut in a *curved* shape (Fig. 2), the anterior and posterior edges will normally describe a curve. (These edges are not under stress, either tension or compression, in this "normally" curved strip of rubber.) The strip of rubber can be deviated laterally without rotation (Fig. 2,*B*). The reason for this is clear. Without variation in tension or compression between the two edges, they both describe the same curve; the mechanism for rotation, illustrated in Figure 1, is not operating.

An S-shaped strip of rubber will behave similarly, as long as there is no inequality in stress between the two edges. If the curved strip is curved further into kyphosis (placed under stress), however, the compression of the anterior edge and the tension in the posterior edge will once more lead to convex-side rotation. Moreover, if the naturally curved strip is extended to any degree, including the position in which it is perfectly straight, then lateral deviation will lead to concave-side rotation.

This rule of behavior may be summarized as follows: The edge under greater tension will describe the straighter line. This holds true in both flexion and extension. If there is no difference in tension between the two edges, there will be no difference in curves, and hence no rotation.

It can now be understood why Rogers was unable to obtain rotation in side-bending of a perfectly round rubber tube, either in flexion or extension. The "edge" of the tube under tension (or compression) could escape through rotation only by placing the next adjacent section of the circumference under exactly the same tension (or compression). Hence there is no escape or advantage in rotation, since the adjacent section of the circumference of the tube would have to take up exactly the same burden. If the rubber tube is interrupted along one edge by multiple cuts, however, or if one edge is in any way thicker, more elastic, or under greater tension than another, rotation will occur in such a manner as to enable the tighter edge to describe the straighter line. If this "tighter" edge of the tube is to one side or the other of the "sagittal" plane, even straight forward flexion or extension, without any lateral deviation, will lead to rotation.

Approaching the problem from another angle, Figure 3-A shows a diagram of a round flexible rod, viewed from above. The arrow shows the direction into which the rod has been deviated to obtain "flexion". There will be agreement that rotation does not occur in this instance. In Figure 3-B, the imaginary planes of reference have been rotated

45 degrees, and hence this rod is now in flexion and lateral deviation in relation to those planes. The attitude of the rod itself has not changed, however, and hence there is still no rotation. Obviously, a round tube or rod *can* be deviated in two planes * without rotation of the middle against the ends.

With these principles in mind, we can study the behavior of the normal spine. The reader will follow the argument more readily if he considers that, as in the rubber model, rotation is due to the *difference in curvature* described by the anterior "edge" (bodies) and posterior "edges" (spines) of the vertebral column.

BEHAVIOR OF THE NORMAL SPINE

A photograph of an isolated spinal column illustrates these principles (Fig. 4). The vertebrae have been joined by two strips of ribbon, fastened with thumbtacks to the tips of the spinous processes and to the middle of the anterior surfaces of the bodies. These two ribbons are intended to represent, approximately, the anterior and posterior soft structures uniting the vertebrae in life. As can be seen, lateral deviation in flexion leads to convex-side rotation (Fig. 5); lateral deviation in extension leads to concave-side rotation (Fig. 6). That this is not due to any property of the articular facets (in favoring convex-side or concave-side rotation, depending upon the degree of flexion) is shown by Figure 7. In this case, when the posterior ribbon is detached, the freed spinous processes escape from its influence and the involved vertebrae do not show convex-side rotation in flexion. Evidently rotation in this model, at least, is controlled solely by the relative tensions in the attached "ligaments", and does not depend in any degree upon the arrangement of the articular facets.

Using an isolated column of vertebral bodies obtained from the cadaver, Lovett noted that lateral deviation in flexion yielded convex-side rotation; in extension, concave-side rotation. This is in accord with the principles already outlined. The behavior of Lovett's model corresponds with that shown in Figures 5, 6, and 7, except that, in straight lateral deviation, concave-side rotation was observed by the author. The anterior longitudinal ligament was apparently tighter (or shrank more in preparation) than the posterior longitudinal ligament.

In attempting to apply his observations to the behavior of the living spine, Lovett did not have the advantage of the roentgen ray. He was compelled to estimate rotation of the vertebrae by observing the behavior of cardboard stickers, pasted on the skin over the spinous processes. These, of course, indicate only the plane of the skin, and are not reliable indicators of actual vertebral rotation. However, Lovett concluded that the living spine behaved in a manner analogous to the models.

In the spine of a living person, the rule still holds that the tighter structure describes the straighter line. However, the situation is more complicated than in the model or cadaver. In the first place, the "ligaments" in the model or cadaver act only at extremes of motion, when they are tense. They are slack and do not influence rotation in any intermediate position. This is not true in the living body, where muscles exhibit tone or elasticity, and hence some resistance to displacement, at any length, even if not actively contracting. In the lumbar spine, for instance, this means that we should expect the posterior structure (the paravertebral muscles) to exert an influence toward convex-side rotation, not only in extreme flexion, but also in every intermediate degree of flexion—and indeed in extension—until overcome, in extreme hyperextension, by the tension which suddenly appears in this position in the anterior longitudinal ligament. Moreover, should some other structure, such as the anterior abdominal muscle wall, intervene to limit hyperextension before the anterior longitudinal ligament becomes tense, concave-side rotation would never occur. This is demonstrated by Rogers' model [4].

* This is a sophism. There is really only one plane if the tube is truly round, since deviation in any plane, or any combination of two planes, is deviation in the direction of a single radius of a circle (viewing the rod from above in cross section). In a perfectly round rod, all such radii are identical.

FIG. 4

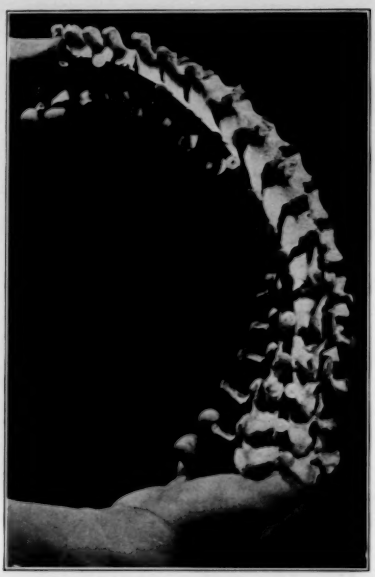

FIG. 5

Fig. 4: Lateral view of column of vertebral bodies.
Fig. 5: Lateral deviation in flexion of a column of vertebral bodies, showing convex-side rotation.

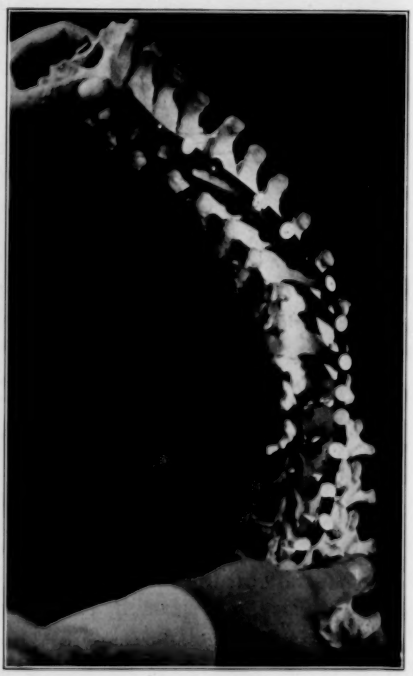

FIG. 6

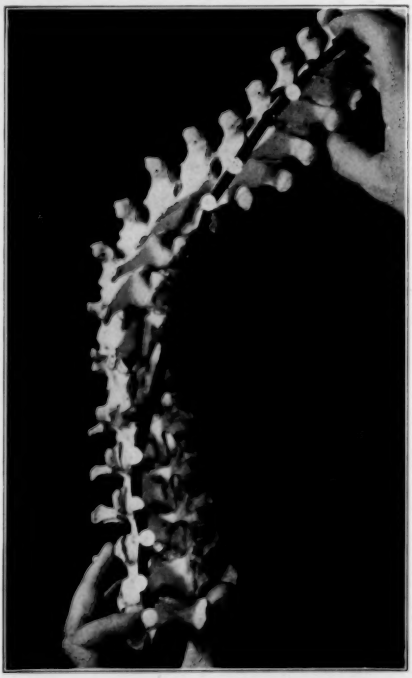

FIG. 7

Fig. 6: Lateral deviation in extension of a column of vertebral bodies, showing concave-side rotation.
Fig. 7: Lateral deviation in flexion of a column of vertebral bodies. Convex-side rotation fails to occur because the posterior ribbon has been detached.

FIG. 8-A

FIG. 8-B

Fig. 8-A: Anteroposterior roentgenogram of spine, with subject supine. Psoas is relaxed.
Fig. 8-B: Anteroposterior roentgenogram of same subject, supine. Left psoas has been tensed by active lifting of left heel off table. Note that the lumbar vertebral bodies are pulled to the left by the psoas, causing rotation and slight curvature to this side.

Moreover, since rotation is influenced by the total tension, and not the tension per unit of cross section, the thick, bulky posterior structures enjoy an additional advantage over the anterior longitudinal ligament. This rotational influence of the posterior structures, furthermore, is magnified by being exerted through a long lever arm, the spinous process.

Apparently the posterior structures have an advantage over the anterior ones which should be reflected in a greater tendency toward convex-side, rather than concave-side, rotation. There are, in addition, *lateral* intrinsic structures, which may exert a significant influence. In Figure 4 the lumbar transverse processes have been marked by black spots. When the soft structures attached to these processes are put under tension, by lateral deviation of the spine, they also attempt to describe a straight line. This, considered alone, will lead to a tendency to convex-side rotation in hyperextension and in normal extension and—to a lesser degree, since the lumbar spine has a smaller range in flexion than in extension—to concave-side rotation in extreme flexion. (This mechanism is much more

FIG. 9-B

FIG. 9-A

Fig. 9-A: T. G. Lateral deviation with subject erect, in neutral position. The middle spinous processes are deviated toward the concavity of the curve, indicating convex-side rotation (of the bodies).

Fig. 9-B: Lateral deviation with subject erect, in hyperextension. Convex-side rotation is noted. (Compare with Fig. 9-A.)

FIG. 10-A

FIG. 10-B

Fig. 10-A: A. C. Control roentgenogram, taken without lateral deviation.
Fig. 10-B: Lateral deviation with subject recumbent, in hyperextension. Note slight concave-side rotation in lumbar spine. (Compare with Fig. 10-A.)

important in scoliosis, where the intertransverse ligament on the convex side is under markedly increased tension, and hypertrophy occurs [1].)

Finally, there are *extrinsic* muscles which can affect rotation. Any structure which can pull the vertebral body or the spinous process to one side or the other (or, alternately, limit their excursion) can contribute to rotation. Figures 8-A and 8-B show this effect of active psoas contraction in pulling the vertebral body to the same side. Obviously all extrinsic muscles which either arise from, or insert into, the bodies or the spinous processes must exert some degree of rotational moment. In symmetrical and relaxed attitudes, this factor does not operate.

Apparently, then, we should expect the behavior of the living spine to differ from that of the model in that convex-side rotation should be dominant in both flexion and extension of the laterally deviated spine. Concave-side rotation could appear only in extreme hyperextension, and only if the anterior longitudinal ligament could be made sufficiently taut,—that is, if the anterior abdominal wall should permit extension to this point.

Rotation in the Living Spine

Fifty-four anteroposterior roentgenograms of the spine were taken in eight living subjects. The roentgenograms of three of these subjects were discarded because of slight curvatures, either alone or in combination with fixed rotation. Thirty-nine anteroposterior roentgenograms were taken of the remaining five subjects, aged twenty-seven, twenty-six, fifteen, eleven, and ten years. All had straight spines without curvature or rotation. Roentgenograms were taken in active and passive lateral deviation in (a) hyperextension, (b) flexion, and (c) neutral position. Some were taken with the subjects sitting, standing, or recumbent, and with the hips flexed or extended. (Not every position was employed in each subject.) Flexion and extension were maintained by applying air pillows under the small of the back or under the pelvis and shoulders, with the patient supine.

For the purposes of this study, "rotation" was considered as rotation of the middle and upper lumbar vertebrae against the sacrum, on the one hand, and against the thoracic spine, on the other hand. In other words, rotation in the segment from the first to the third lumbar vertebrae, as measured by the position of their dorsal spinous processes, was considered to be valid only if both the segment above and the segment below were rotated in an opposite direction.

The results may be summarized as follows: In the adults, convex-side rotation appeared when the spine was in lateral deviation, whether in flexion, extension, or neutral position (Figs. 9-A and 9-B). The tendency to convex-side rotation was strongest in flexion and weakest in hyperextension. It was stronger in one subject than in the other, in whom very slight, if any, convex-side rotation was noted in extension, although convex-side rotation was well marked in flexion. Concave-side rotation could not be obtained, although roentgenograms were taken in active and passive hyperextension, with the subjects sitting, standing, and supine.

The children showed less tendency to rotation with the spine in lateral deviation in either flexion, extension, or neutral position. Slight convex-side rotation, or most frequently none at all, appeared in all but one roentgenogram. However, in one subject aged fifteen, a roentgenogram taken with the subject in passive lateral deviation in extreme hyperextension (Fig. 10-B) revealed slight *concave*-side rotation. This was the only film of the thirty-nine in which concave-side rotation was visible.

DISCUSSION

There has been a good deal of discussion about the occurrence of concave-side rotation in the living spine. The opinions of Lovett and of Schulthess in favor and those of Werndorff and Reiner in opposition have been summarized by Steindler in his excellent discussion of the subject. Steindler concluded that ". . . concave side rotation is not the expression of a natural tendency, but must be the result of some active muscular effort. . .". The analysis and findings reported in this paper would seem to confirm Steindler's statement. (The effect of active psoas contraction can be seen in Figures 8-A and 8-B. It is to be noted that, if the intertransverse muscles on one side of a normally lordotic lumbar spine are in spasm, they tend to produce a curve concave to the affected side, with *concave*-side rotation.)

Concave-side rotation plays no part in clinical structural scoliosis. On the other hand, one of the component forces contributing to the convex-side rotation seen in scoliosis is produced by the soft-tissue mechanism described in this paper,—the tendency of structures under tension to assume a straighter line.

CONCLUSIONS

1. The intrinsic mechanism for rotation in combination with lateral deviation in the normal spine depends upon soft-tissue tensions rather than upon the arrangement of the

spinal articular facets. The behavior may be summarized by stating that the structure under the greater tension will describe the straighter line.

2. Thirty-nine roentgenograms of five subjects showed a tendency to convex-side rotation in both flexion and extension of the laterally deviated living spine.

REFERENCES

1. Arkin, A. M.: The Mechanism of the Structural Changes in Scoliosis. J. Bone and Joint Surg., **31-A**: 519–527, July 1949.
2. Lovett, R. W.: The Mechanism of the Normal Spine and Its Relation to Scoliosis. Boston Med. and Surg. J., **153**: 349–358, 1905.
3. Ober, F. R., and Brewster, A. H.: Lovett's Lateral Curvature of the Spine and Round Shoulders, Ed. 5. Philadelphia, P. Blakiston's Son and Co., 1931.
4. Rogers, S. P.: Mechanics of Scoliosis. Arch. Surg., **26**: 962–980, 1933.
5. Steindler, Arthur: Diseases and Deformities of the Spine and Thorax, p. 144. St. Louis, C. V. Mosby Co., 1929.

TREATMENT OF THE "HOPELESS" CEREBRAL-PALSY PATIENT

BY HERBERT E. HIPPS, M.D., WACO, TEXAS

Many "hopelessly paralyzed" cerebral-palsy patients have educable minds and can be rehabilitated to a degree well worth the effort. By "hopelessly paralyzed" is meant the individual, afflicted with cerebral palsy, who cannot hold his head up, sit alone, or talk, and who has no control, or effective use, of his hands.

GENERAL PLAN OF TREATMENT

Most cerebral-palsy patients, particularly the group under consideration, learn motor activities very slowly; hence, they should be taught only one thing at a time. For those severely involved, a training program of muscle relaxation, coordination, reciprocation, speech, voluntary use of the hands, voluntary movements of the legs, standing, and walking, all taught at the same time, presents an insurmountable obstacle. It is possible often for this type of patient to learn, by continuous, unrelenting effort on one thing only.

Some of the motor skills acquired by normal individuals are listed (Table I). In Table II these motor skills are arranged in the order of their importance for the development of a self-sustaining individual. They should be learned in this order.

Head balance and voluntary control of the head have been placed first, for two rea-

TABLE I

Motor Skills of a Normal Individual

Balancing head in erect position	Pointing
Voluntary control of head movements	Moving an object in a purposeful manner
Sitting balance (sitting alone)	Feeding self
Standing balance (standing alone)	Combing hair
Walking	Brushing teeth
Running	Dressing self
Bladder and bowel control	Writing
Climbing steps, ladders, et cetera	Games
Dancing and athletics	Typewriting and use of business machines
Holding up arms	Playing musical instruments
Grasping and relinquishing objects	Juggling
Throwing	Sleight-of-hand, contortionist skills, et cetera

sons: These are the simplest and most elementary of motor skills—among the first things a normal baby learns—and they must be learned before he can be expected to sit alone. Furthermore, sitting and moving the head in various directions promote mental development, as these activities extend the patient's visual horizon from the ceiling to the world about him.

Next in importance is speech, followed closely by the use of one or both hands. After some degree of skill has been attained in these activities, sitting balance can be learned and, later, standing balance; then walking is attempted.

In each instance only one of these activities is learned at a time. When the individual has learned one of them, another is added, and he continually practises both of them. When he has learned the second, a third is added, and he continually practises all three. This is true throughout the list, with one exception; this exception occurs after a child has learned voluntarily to control head movement. Then he can be taught speech and the use of his hands at the same time, since these two motor activities augment each other.

This phase of training—that is, learning speech and the use of the hands—is exceedingly long and arduous, since these are the most important self-sustaining activities. Time and effort spent in trying to teach the child to walk, when he cannot balance his head, speak, or use his hands, are wasted.

Teaching Head Balance

Two things are wrong with the neck muscles of the cerebral-palsy patient: (1) He has no voluntary control over them. (2) The muscles are so weak, because he has spent all of his life lying in bed, that, even if he did have full voluntary control over them, he could not hold his head up and move it for very long. His head, therefore, must be supported in such a way that it can be moved voluntarily. A specially devised chair-brace will perform this function (Fig. 1).

The patient is placed in this chair, with one lower extremity on each side of the mid-seat post. The post is movable, and it is adjusted to fit snugly against the crotch. Straps across the chest and pelvis, across the lap, and over the mid-portions of the legs, hold the child securely and comfortably in position. His head is loosely supported in an upright position, in a supporting device which is movable, permitting free rotation of the head.

The mother may put this chair in the kitchen in such a place that, as she moves back and forth, the child must turn his head to watch her. The longer the child is kept in the chair each day, the more rapidly will he learn, if he is at all capable of learning. The more often he moves his head from one side to the other, the more rapidly will he learn to control movements of his head, and the more rapidly will the muscles of his neck gain strength.

FIG. 1

Chair-brace for teaching head balance and sitting balance.

TABLE II

MOTOR SKILLS IN ORDER OF IMPORTANCE

Balancing head in erect position
Voluntary control of head movements in erect position
Use of arms, hands, and fingers
Speech
Bladder and bowel control
Sitting balance (sitting alone)
Standing balance (standing alone)
Walking

During this time the child should not be given any other form of therapy. All of his waking hours should be devoted to learning to balance his head and to move it voluntarily in any direction.

After the child has learned to rotate his head easily from side to side, the head support gradually may be left off. At first it may be removed for only an hour in the morning; then, in a few weeks or months, for an additional hour in the afternoon. These intervals are gradually increased, until finally he is sitting in the chair all day, balancing his head and moving it in any direction without support. At that point, the head support may be removed from the chair. The time has come to teach him to use his hands and arms, and to learn speech.

Teaching Speech and Use of Hands

Speech is best taught by a trained speech therapist. The mother can, for the most part, teach the child to use his hands and arms, but she should seek instruction from a competent physical therapist at intervals.

A child is much more eager to play with a moving toy than with one that is lying still. Toys, therefore, are suspended from the overhead arm, built for that purpose. The mother must make frequent attempts during the day to get the child to play with these toys.

At no time is the child given exercises for relaxation, coordination, or reciprocal motion of the arms, stretching or massage, or passive movement. Any time and effort spent in giving such exercises are wasted; it is far better to try to get the child to perform some purposeful movement himself.

These children learn best directly, by doing. If a normal child is to learn to play a piano, he practises on the piano; he learns by constant, repetitious efforts, by trying and working, again and again. This is also the most certain way for the child with cerebral palsy to learn to do any one thing.

Most of the "hopelessly" involved must stay in this second stage of training for an exceedingly long period of time. However, if they learn enough speech to make themselves understood, and learn to use the hands well enough to feed themselves, brush their teeth, comb their hair, and perform some of the other more necessary motor activities of normal living, then the next stage can be started,—sitting balance.

Teaching Sitting Balance

This same special chair-brace may be used in teaching the child to balance himself in a sitting position. The chest strap is removed and the back of the chair, which is adjustable, is lowered to about the middle of the chest. When the child has learned to balance the upper portion of his body, the back of the chair is lowered to the level of the waist, and still later to the level of the pelvis. Finally the pelvic strap is removed, and the back of the chair is dropped below the level of the seat. This leaves the child sitting on a stool with side arms. After this, or coincident with the lowering of the back piece, the side arms are gradually moved away from his body, and finally they are removed.

TABLE III

RESULTS IN NINETEEN PATIENTS

Patient	Type of Paralysis	Age at Start of Treatment (*Years*)	Time Required to Learn Head Balance (*Months*)	Time Required to Learn Sitting Balance (*Months*)	Improved Hand Usage
M. J.	Spastic	3	5	3	Yes
T. S.	Spastic	2	1	2	Yes
S. T.	Spastic	4	Not yet	Not yet	No
A. J.	Spastic	6	7	12	Yes
S. M.	Spastic	1	3	6	Yes
M. M.	Spastic	4½	9	18	Yes
J. C.	Spastic	3	4	8	Yes
A. S.	Spastic	5	13	Not yet	No
E. I.	Spastic	2	6	4	Yes
K. J.	Spastic	2½	2	1	Yes
M. W.	Spastic	5	Not yet	Not yet	No
G. S.	Ataxic?	3	23	Not yet	No
P. S.	Ataxic?	3½	Not yet	Not yet	No
R. B.	Athetoid	2	12	20	No
J. S.	Athetoid	17	Not yet	Not yet	No
L. T.	Athetoid	9	Not yet	Not yet	No
D. D.	Athetoid	8	8	6	Yes
S. K.	Athetoid	2	1	3	Yes
T. M.	Athetoid	12	10	8	Yes

Seldom does one of these severely afflicted patients progress beyond this to the stage of learning to stand alone and to walk; but, even if he does not, he has by this time become a self-sustaining individual.

COMMENT

Nineteen "hopeless" patients have been treated in this way. The results (Table III) are not spectacular, but it must be remembered that we are dealing with a group of patients who have, for the most part, been considered as hopeless invalids. Most of them already had been treated for considerable periods of time by the usual methods of treatment and had shown no improvement. This new and common-sense approach to the treatment and training of these patients is yielding slow, but gratifying, results.

LATERAL ROENTGENOGRAPHIC EXAMINATION OF THE THORACIC SPINE *

BY DR. GERALDO GUERREIRO, SÃO PAULO, BRASIL

The roentgenographic study of the thoracic portion of the spinal column has offered a number of serious problems, especially when the films are taken with the spine in the profile position. Oblique roentgenograms taken from various positions have been employed in an effort to achieve a satisfactory examination; but even with the use of this method, the small compression fractures can pass unperceived. Similarly, arthritic changes or destructive developments in the posterior portion of the body of the vertebra and in the articular process may also go unnoticed.

FIG. 1

FIG. 2

In order that the difficulty might be overcome, a roentgenogram was taken of the spinal column in profile, the patient being placed in an orthostatic position with vertebral support. The patient was then told to breathe slowly and deeply during the course of the roentgenographic exposure. (Fifty kilovolts and fifteen milliamperes were used for a period of fifteen seconds; there was a focal distance of two meters.)

As a result of this technique, there was a complete disappearance of the ribs from the plate. The spinal column was outlined with the clarity of the actual anatomical structure (Figs. 1 and 2).

Another interesting result was that the aortic arc also appeared to be clearly outlined.

* Read at the First Brazilian Radiology Congress, São Paulo, Brasil, September 1948.

OSTEOMALACIA* †

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Legend and history, so difficult to separate, have contributed a number of ancient stories of patients who suffered from softened bones and resultant deformities. There are several notes of reports by early Arabian physicians, one of whom described a case which was seen in the days of Mohammed [27, 33]! Throughout succeeding centuries there appeared occasional references to such disorders, which were usually termed mollities ossium ("softening of the bones") or malacosteon ("softening of the ribs"). It is not known precisely when the term osteomalacia came into use or by whom it was first applied, but all three words began to be used interchangeably in the description of any disorder which was accompanied by skeletal softening or deformity.

Early in the eighteenth century it was noted that such skeletal disease was prevalent among adult multiparous females, and that the resultant pelvic deformities frequently prevented normal deliveries. In fact, as early as 1768, Caesarean section was being performed in consequence [52]. The disease, known appropriately as "back and thigh pains", began to be recognized as a specific type of bone softening, and in 1772 Levacher de la Feutrie introduced the idea that it was adult rickets. He was unwilling to assume that it was identical with childhood rickets, as were later pathologists such as Lobstein. In the meantime Schütte had reported, in 1824, on the curative value of cod-liver oil. He referred to five cases seen as far back as 1800, four of which were rickets. The other patient, a woman stated to have chronic rheumatism with paralysis of the lower extremities, was probably suffering from osteomalacia. All were cured by taking cod-liver oil [68]. These observations were not lost on Trousseau and Lasègue, who not only supported the theory that rickets and osteomalacia are the same disease, but reported several cases of advanced osteomalacia cured by the administration of cod-liver oil.

Unfortunately the thinking of these men made little impression on their colleagues, who, believing that rickets and osteomalacia were entirely separate diseases, began to search indiscriminately for the cause of the latter. A severe blow to the theories of Trousseau and Lasègue was delivered by Virchow who, in 1853, stated categorically that there was no similarity between rickets and osteomalacia. The final state of confusion was produced by the persistent tendency of most physicians to call any bone-destroying lesion osteomalacia, regardless of the etiology. Occasionally someone was moved to wonder, as Bartlett did, whether the patient's fungating breast cancer might perhaps be concerned, or whether "osteomalacia pthisica" might differ from "osteomalacia carcinomatosa". It is interesting that Bence-Jones' original description of the urinary protein known by his name was reported under the title: "On a New Substance Occurring in the Urine of a Patient with Mollities Ossium". The postmortem description of the gross appearance of the bones has been widely quoted as sufficiently characteristic to be diagnostic of osteomalacia!

Virchow and his followers, having promulgated the "halisteresis" theory, built an elaborate explanation. The difference between rickets and osteomalacia, Virchow said, was that, in the former, osteoid tissue was laid down and failed to calcify; while in the latter, the mineral content of mature bone was actually leached out. Such a theory necessitated the postulation of a leaching substance and, since one decalcifies bone *in vitro* by means of acids, the hunt was on for an acid which could circulate in the blood and attack the bone. A few investigators, such as Moers and Muck and Otto Weber, claimed to have found abnormal amounts of lactic acid in the bones and urine of patients with osteomalacia. Subsequent reports failed to confirm this work [36, 49].

* Received for publication February 28, 1949.
† This work was aided by a grant from the Douglas Smith Foundation.

The picture began to be clarified again when Pommer, in 1885, took issue with Virchow. Pommer re-examined the evidence and concluded that rickets and osteomalacia were identical. Some years later von Recklinghausen aligned himself with Virchow, but it was not long before such eminent men as Cohnheim, Schmorl, and Looser agreed with Pommer. Although there are still a few who object, it is now quite generally accepted that the pathology and etiology of rickets and osteomalacia are the same.

In the meantime the clinicians, with a fine disregard for all that had gone before, also were searching for the cause of osteomalacia. In 1879, Fochier reported spectacular improvement in an osteomalacic woman upon whom he had performed a panhysterectomy, following a Caesarean section necessitated by pelvic deformity. Since osteomalacia had always been a disease predominantly of females, this report aroused great enthusiasm and oophorectomy suddenly became a very popular operation, in spite of the fact that no one could demonstrate any pathological changes in the ovaries removed. After the initial enthusiasm had worn off, it became apparent that the percentage of cures was not so high as had been thought. When methods of studying the calcium metabolism became available, it was soon shown that any benefit from such surgical treatment probably resulted from the prevention of further pregnancies.

By one or another investigator, almost every gland of internal secretion has been considered responsible for osteomalacia. Scipiades announced, on the basis of animal experimentation, that the true cause of osteomalacia was thymus deficiency. He reported a case in which the patient's symptoms had begun during her eighth pregnancy and had progressed for two years. Scipiades transplanted the thymus gland of a newborn infant to the abdominal muscle of the patient. The woman is said to have improved rapidly and to have remained well for three years. There seems to have been little follow-up on this work.

In 1905, Hoennicke, who had noted the occasional occurrence of thyroid dysfunction and osteomalacia in the same patient, advocated thyroidectomy as a therapeutic measure. About the same time Cristofoletti decided that a hypofunction of the adrenals (secondary to deranged ovarian activity) was at fault, and began to treat his patients by castration plus adrenalin injections. Treatment with adrenalin had quite a vogue thereafter, but was gradually discarded. Arcangeli and others were, in the meantime, supporting the theory that osteomalacia was an infectious disease, due to a specific diplococcus [3, 34]. Their researches led them to abandon treatment by means of prolonged administration of chloroform and to use autogenous vaccines instead.

In 1906, Erdheim reported the occasional hypertrophy or hyperplasia of the parathyroid glands in cases of osteomalacia. He and several of his colleagues rightly interpreted this finding as indicating a secondary response to underlying skeletal disease. However, many clinicians failed to understand the conclusions, and thus parathyroidectomy for osteomalacia had a brief period of popularity.

Nor was the pituitary gland overlooked. In 1911, Bab decided that a normal-appearing ovary might still be deficient in function as a result of derangement of the hypophysis. Since a hyperfunctioning hypophysis produced acromegaly, a condition which may be attended by amenorrhoea, Bab treated osteomalacia with hypophyseal extracts. The results, as might be expected, were negligible.

It remained for Curschmann, Naegeli, and others to cover the entire field with a theory that not one, but all, glands of internal secretion might be deranged; they, therefore, advanced the theory that osteomalacia was a "pluriglandular upset".

The period of confusion began to end when the admirable pathological studies of Pommer, Looser, and others could be supplemented with metabolic investigations. The pioneers in this field were a group of Boston investigators [23], whose first studies were made on a sixteen-year-old girl, said to have been crippled for several years with osteomalacia. This history, together with the fact that the preponderance of the calcium lost by this patient appeared in the urine, suggests that the condition was already in a remission. How-

ever, the studies were of considerable value, since they were repeated twice after the patient had undergone castration [47]. The second experiment, performed a few months after operation when there was clinical improvement, showed a markedly positive calcium balance, an increase in nitrogen retention, and a reduction of sulphur retention, which had previously been abnormally high. One year after operation the disease process again became acute and the experiments were repeated. By this time the loss of calcium was even greater than it had been initially, and most of the calcium was found in the faeces. The conclusion was unavoidable that castration had no permanent effect on calcium metabolism and that its apparent beneficial effect on osteomalacia must be in preventing pregnancy [47]. This conclusion was supported further by subsequent work on castrated dogs, both male and female.

McCrudden also furnished telling evidence against the halisteresis theory with his chemical analysis of osteomalacic bones, both animal and human [46, 48]. In both he found, as others had, that the total ash content, in particular the calcium and phosphorus, was markedly decreased. Inorganic sulphur and magnesium were, in contrast, increased to four or five times normal values. Such a variation in composition could not result from simple removal of calcium and phosphorus from mature bone, but supports the hypothesis that the old bone is removed *in toto* and a new material, rich in sulphur and magnesium, is laid down in its place.

The most significant modern study of osteomalacia has been done by the workers at the Peiping Union Medical School. In 1925, they published the first of a series of excellent metabolic studies, which cover most phases of the problem. That they were dealing with the same entity which was discussed by other workers is proved by their careful supplementary pathological studies and the chemical analysis of bones [9, 10, 26, 38–42, 53].

Their findings, in brief, are as follows: Patients with active osteomalacia are always in negative calcium balance. In the milder cases, normal levels of serum calcium and phosphorus are exhibited. In the more severe cases, where serum calcium may be low (values of 4 milligrams per 100 cubic centimeters have been recorded), the parathyroids are usually stimulated and the serum phosphorus falls. In any case, the urinary calcium is markedly decreased or completely absent which *ipso facto* disproves the idea that there is a continuous "endogenous excretion", such as would be demanded by the halisteresis theory. Furthermore, such patients derive no benefit from increased calcium intake by mouth, but simply lose more calcium in the stools. If the intake is increased by intravenous administration (in the absence of other therapy), there is calcium retention, the clinical symptoms subside, and there is evidence of skeletal repair. Therefore, the primary defect is obviously lack of absorption of calcium from the intestinal tract. When these patients are given vitamin D or ultraviolet light (which stimulates the skin to form vitamin D), these phenomena are abolished. There is increased absorption, so that the faeces contain less calcium and eventually some is excreted in the urine. The patient maintains a positive calcium balance, and this state of affairs continues for eight to twelve weeks after the cessation of therapy. The maximum effect of vitamin D seems to be achieved with ordinary doses, and the administration of increased amounts produces no additional changes.

The studies on the effects of pregnancy and lactation produced most interesting results. Pregnancy does have a deleterious effect on calcium retention, so that in a borderline patient it may precipitate active osteomalacia. Most patients, however, when questioned carefully, admit that symptoms began after delivery. Analysis of stillborn infants shows that the total calcium loss during pregnancy is not more than 23 grams, most of which is deposited in the last trimester. During lactation, however, the daily loss may be as much as 250 to 350 milligrams. Since the total calcium in the adult human female body is somewhere around 500 to 700 grams, a woman can lose one-tenth of her total body calcium during six months of lactation.

The final study in this series showed that the calcium metabolism of the nursing in-

fant fluctuates in relation to that of the mother. The defects in both can be eliminated by treatment of the mother.

The type of deficiency osteomalacia studied by these workers has become a disease of great rarity in the Western world, where diet and hygiene are on a fairly high level. It is seen commonly only in certain parts of India and China, and the reasons for this have been worked out by several investigators. In 1916, Agnes Scott published her careful observations on osteomalacia in India. The most interesting conclusion was that the distribution of the disease is not closely related to any factor except social custom. Thus the disease is found predominantly in Mohammedan and high-caste Hindu women who, from the time they are married at ten or twelve, seldom again go outdoors. Their houses are built so that sunlight almost never enters. The men and the lower-class women who work outside do not have osteomalacia. These observations have since been confirmed by others [31]. In China, osteomalacia is limited to a few areas in the north, where the disease is extremely common. In these areas the same factors of seclusion seem to be augmented by a more selectively deficient diet.

In the United States and Canada, osteomalacia has always been considered an exceedingly rare disease. Actually it is less common than the number of published reports would indicate, for the same confusion as to terminology has complicated even the modern literature. Many reports, such as those of Wheaton, Russell, Olin, Holmes, and Hadley, contain so little data that it is impossible even to guess what the patients might have had. Many others are sufficiently detailed so that, with the wisdom of hindsight, we can recognize a description of osteogenesis imperfecta [1, 16], metastatic malignancy [61, 72], multiple osteomyelitis [33, 56], rheumatoid arthritis [35], or even congenital deformities [51]. There is also a group of papers which describe florid rickets as osteomalacia [8, 32]. However, true osteomalacia does occur on this Continent, and now that accurate methods of diagnosis are at hand, more cases will undoubtedly be found.

WHAT IS OSTEOMALACIA?

As Albright and his associates have pointed out, the term osteomalacia should be reserved to describe any disorder of adults which results in the presence in the bones of abnormal amounts of osteoid tissue. This is to say that the proof of diagnosis necessarily rests upon the microscopic examination of bone. In the absence of detailed metabolic data, such is certainly the case, although the clinical studies, the roentgenograms, and the response to therapy may afford very suggestive evidence.

Any condition which causes the body's calcium stores to be depleted sufficiently can cause osteomalacia. Such depletion can come about either through insufficient absorption of calcium or through excessive loss. The following discussion of the possible etiologies of osteomalacia has been modified from Albright's classification:

A. *Insufficient Vitamin D*

1. *Simple Lack:* This is, of course, rare in the United States, where the average adult diet contains sufficient vitamin D and the dietary intake is amply supplemented by the effect of sunshine. In this country this type of deficiency is usually confined to persons who, for one or another reason, lead an abnormal life [14]. It is a voluntarily imposed abnormal life, as has been mentioned, which makes this type of osteomalacia so common in parts of India and China. There is no well substantiated report of such a case occurring in North America. Even the patient so well studied by Gargill, Gilligan, and Blumgart, who responded dramatically to the administration of cod-liver oil and ultraviolet light, is probably not a true example. Albright restudied the same patient in 1945 and found a daily urinary-calcium excretion of 200 milligrams, no matter what the intake. When the intake of calcium covered this daily loss, the patient did well even without vitamin D. Albright concluded that this case probably belongs in Group C (Idiopathic Hypercalcuria).

As a result of dietary restrictions experienced during and after the first World War, numerous cases of a painful bone disorder developed in the Central European countries. Adequate correlated metabolic and pathological studies were of course impossible at that time, but many of these cases of "hunger osteopathy" responded well to the administration of cod-liver oil. Almost certainly these were instances of osteomalacia due to lack of vitamin D [15, 30].

2. *In Gastro-Intestinal Disorders:* Vitamin D, being fat-soluble, is not absorbed in diseases such as sprue and steatorrhoea. Furthermore, the attendant diarrhoea, which results in rapid passage of the intestinal contents, adds to the difficulty of calcium absorption. The development of osteomalacia in such cases is not at all uncommon [57, 63, 71].

A similar effect can be observed in certain patients who are addicted to laxatives, especially oils and those mineral salts which tend to form insoluble calcium compounds in the gut [59]. As might be expected, the same state is occasionally established in patients who have long-standing achlorhydria with reduction of calcium absorption [59].

B. *Renal Acidosis*

1. *Tubular Insufficiency without Glomerular Insufficiency:* It is this category of cases which Albright and his co-workers have recently clarified. Patients with this disorder have a decreased ability to excrete acid radicals. Therefore, whenever an added strain is placed upon the tubules (as occurs with increased intake of acid radicals, for example), the kidney has to have added base. Since the only depot of readily mobilized base is the skeleton, the bones tend to become depleted as long as the abnormal balance lasts. Furthermore, ingested calcium is insufficient to cover the loss, so that new bone matrix, although laid down in the usual amounts, tends not to become calcified. A complete explanation of this phenomenon, together with the experimental evidence, can be found in Albright's admirable paper on osteomalacia and late rickets.

2. *Fanconi Syndrome:* In cases of this rare congenital defect, osteomalacia is only one of the clinical and pathological manifestations. Most investigators now believe that this peculiar syndrome depends upon a dysfunction of the kidney tubules. There is usually present, in addition to the phenomena described in the preceding section, glycosuria, albuminuria, and an increased concentration of amino acids in the urine. Although there is no perfect agreement, it is generally assumed that the constant acidosis is responsible for the development of defective calcification of osteoid which, of course, does not respond to ordinary antirachitic therapy. Azotaemia is usually not present until late in the disease, when secondary kidney damage has occurred. By this time, when there is retention of phosphorus together with hypocalcaemia, there is stimulation of the parathyroid glands and the picture of secondary hyperparathyroidism is superimposed.

Symptoms of this disorder usually appear within the first two years of life and progress rapidly until death. For a detailed description of this syndrome and its manifold variations, the paper of McCune, Mason, and Clarke should be consulted.

C. *Idiopathic Hypercalcuria*

Apparently a low kidney threshold for calcium develops in some patients. They excrete continuously a large amount of calcium in the urine, no matter how depleted their skeleton may become. It is to this class of patients that Case 1 belongs.

Albright and his associates included in their classification a fourth group which comprises the transient osteomalacia which may be seen immediately following removal of a parathyroid adenoma. With the sudden fall in serum calcium, the mineral balance is sufficiently deranged so that the processes of calcification are temporarily interrupted. Since this state is temporary, self-limited, and of no clinical significance, it seems preferable to mention it as a matter of academic interest rather than as a variety of true osteomalacia.

As is readily seen from the preceding discussion, the state of osteomalacia has a variety

of basic causes. Obviously, therefore, no routine treatment can be given. In each case therapy must be directed toward the underlying defect, and this therapy must be supplemented by adequate replacement of calcium. Even when primary lack of vitamin D is not a factor, this substance is generally useful in increasing the absorption of calcium from the intestinal tract.

INCIDENCE OF OSTEOMALACIA IN NORTH AMERICA

There are available sixty-five papers, reputedly reporting some ninety cases of osteomalacia from the United States and Canada. If one excepts the cases reported by Albright and his associates, there are among all the other papers reports of two proved cases, only one of which had pathological examination. Six other cases had suggestive histories and findings, but the evidence was insufficient for proof.

On the other hand, the fact that Albright has been able to collect six unquestionable cases and three others in which the conditions for the development of osteomalacia were certainly present leads one to suppose that the disorder is not so much rare as it is undiagnosed. Such was the case with the patient who prompted this report.

CASE 1. This man was born and had lived all his life in a small town in Indiana. His mother had died at forty-six years of "myositis", but his father was still living and well. There were twelve living siblings and one who had been a war casualty. The family had all lived together under the same conditions, and there was no history of even minor dietary abnormalities.

The patient was said to have had pertussis, measles, mumps, chicken pox, smallpox, pneumonia, and diphtheria in childhood, but from the age of fifteen until he was thirty-seven he had had no illnesses or accidents and had worked as a farmer. At that time he was five feet, four inches tall, and weighed 130 pounds.

During the winter of 1936, when he was thirty-seven years old, the patient first noted soreness and stiffness of the lower portion of his back. His local physician could find nothing to account for these symptoms. Roentgenograms revealed a normal lumbosacral spine. The soreness, however, gradually progressed up the spine and then to all four extremities. Bone tenderness appeared, and it was increasingly difficult for the patient to get up out of a chair because of stiffness and soreness of the hips. One year after the onset the patient was examined by another physician. At this time the man was only five feet, three inches tall, and he weighed 112 pounds. He had tenderness of many bones, but especially over the right eleventh rib (probably a pathological fracture). His blood counts and roentgenograms were reported as normal and he was treated for arthritis.

Two years later, when the patient entered a diagnostic center, his condition was still worse. Roentgenograms showed moderate osteoporosis with compression of the bodies of the eighth, ninth, and tenth thoracic vertebrae, and a pathological fracture of the right eleventh rib. General laboratory studies were normal, but the serum calcium was 10.4 milligrams per 100 cubic centimeters, the phosphorus was 1.9 milligrams per 100 cubic centimeters, and the phosphatase was 6.8 Bodansky units. The diagnosis was ruptured intervetebral disc together with muscular dystrophy.

FIG. 1-A

Case 1. Nine years after onset of symptoms. Note marked deformity of chest and extremities and extreme muscle atrophy.

By 1940, when he was seen at another medical center (three and one-half years after the onset of symptoms), he was only five feet, one inch tall, and was unable to stand or walk. To the previous findings had been added marked bone tenderness, atrophy of muscles, and beginning contractures of joints. Roentgenograms were reported as showing "marked generalized osteoporosis". Blood studies still showed a serum-calcium level of 9.5 milligrams, phosphorus 1.6 milligrams, and phosphatase 6.4 Bodansky units. Because

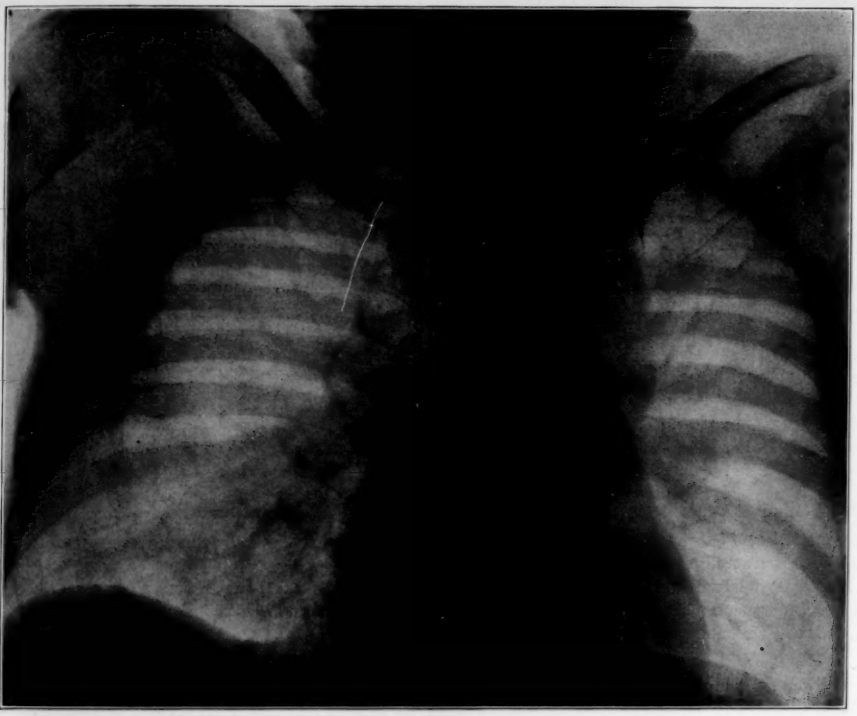

FIG. 1-B

Roentgenogram of chest shows that density of the bone is reduced almost to that of the soft tissue. There have been fractures of most of the ribs and the base of each acromion process.

FIG. 1-C

Roentgenogram of wrist and hand shows similar changes. There is no disorder of any of the joints.

muscle biopsy was reported as showing atrophy and fibrosis, a diagnosis of muscular dystrophy or myositis fibrosa was made.

During the next four years the patient remained bedridden at home. He had numerous fractures which healed with deformity. Joint contractures gradually increased and the "soreness of bones" became so severe that the patient was unable to move enough to feed himself.

When he first appeared at the University of Chicago clinics in 1945, nine years after his symptoms had begun, this man was five feet tall and weighed seventy-two pounds. The marked skeletal deformities, joint contractures, and muscle atrophy are shown in Figure 1-A. He had very active tendon reflexes, and fibrillations of the muscles were evident for several seconds following contraction. There were no sensory changes. All bones were exquisitely tender and the slightest motion caused intolerable pain. There was no evidence of cataract, and his teeth were in good condition.

Roentgenograms showed a spectacular degree of osteoporosis of all bones of the trunk and extremities with multiple healed fractures. Also evident were symmetrical "pseudofractures" in ribs, clavicles, and at the base of each acromion process (Fig. 1-B). The skull appeared relatively normal and there was no evidence of primary joint involvement.

Significant laboratory data included: serum calcium 10 milligrams and phosphorus 1.7 milligrams per 100 cubic centimeters; alkaline phosphatase 17.4 (normal by the method used is 1 to 4); proteins 7.14 milligrams per 100 cubic centimeters with normal albumin-globulin ratio. The urine maintained a slightly acid pH. Other initial values were: blood urea 33 milligrams per 100 cubic centimeters; carbon dioxide 27 millimoles per liter; serum pH 7.45; cholesterol esters 143.4 milligrams per 100 cubic centimeters; chlorides 99.8 millimoles per liter; oral glucose-tolerance test normal; and basal metabolic rate plus 7 to minus 9 (Benedict-Roth method). The phosphorus and calcium clearance test was normal. Routine blood counts and serology were normal (as they had always been).

During the first few days of his hospital stay the patient was on a regular diet and his only medications were small amounts of aspirin, codeine, and seconal. On the ninth day he had two episodes of what appeared to be major Jacksonian convulsions, not accompanied by a cry or by carpopedal spasm, although the latter was hard to judge because of his deformities. The two spells occurred within two hours of each other, lasted ten minutes each, subsided without treatment, and did not recur. No such episode had occurred before. On this day the blood calcium was 11.8 milligrams and the phosphorus was 1.65 milligrams per 100 cubic centimeters. An electro-encephalogram was reported as showing "either an idiopathic epilepsy or some condition which causes generalized irritation".

A few days later a rib biopsy was performed under local anaesthesia. The sections show a spectacular picture. The hypertrophic marrow and the trabecular pattern are normal. However, very little of the bone matrix is calcified, and the great mass is pure osteoid tissue (Fig. 1-D).

The diagnosis was thus established as true osteomalacia, or adult rickets.

Studies of this man's urine showed that he was excreting between 45 and 65 milligrams of calcium each twenty-four hours. In the presence of marked skeletal depletion, this fact is evidence that the primary defect was not lack of absorption of calcium from the intestinal tract. This assumption is further supported by the fact that, after one month of a high-calcium diet (approximately 2 grams daily), the urinary calcium was between 80 and 100 milligrams per twenty-four hours. When 150,000 units of vitamin D was added to this regimen, the urinary calcium rose to 331 milligrams which suggests that, although absorption was probably increased, retention was not increased proportionately. The absence of values for faecal calcium precludes the calculation of calcium balances. During this time there was no significant change in the serum chemistry.

Further studies of this nature were impossible, because on August 20, seven weeks after admission and sixteen days after therapy had been initiated, the patient died very suddenly.

Postmortem examination revealed the generalized arteriosclerosis and coronary occlusion which were the cause of death, and the extensive skeletal changes. Of special interest were several rib fractures which had united by callus that was completely composed of osteoid tissue (Fig. 1-E). The parathyroids were described as normal in appearance. Unfortunately, they were lost, so that microscopic examination was not possible.

It is believed that this man represents an instance of "idiopathic hypercalcuria". For some reason a low renal threshold for calcium developed, so that, even when his bones had been almost completely replaced by osteoid tissue, he did not retain enough minerals to allow calcification. Since this state of affairs had existed for nine years, it is remarkable that there was never a tendency for the formation of urinary stones. Presumably the condition might have been alleviated if the patient had had appropriate treatment for a long enough period.

CASE 2. The second patient was well until he was twenty-four years old; at that time diarrhoea developed. Two or three times each month he would have acute attacks during which he passed from five to ten loose, foul-smelling stools daily. Between exacerbations he had three or four bulky, poorly formed stools each day.

FIG. 1-D

Photomicrograph (× 100). The darker-staining portions of the trabeculae, which are bone, are bordered on all free surfaces by broad bands of lighter-staining osteoid tissue.

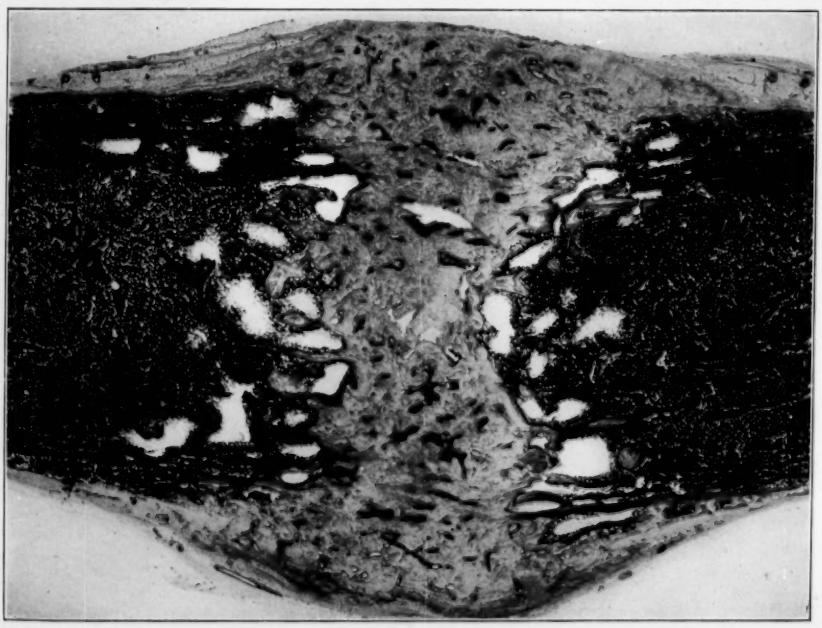

FIG. 1-E

Photomicrograph (× 7.5) of rib fracture. The fracture has united by abundant callus which is entirely composed of osteoid tissue. Note atrophic bony trabeculae on both sides of fracture.

Gradually he lost weight, became pale and generally weak, and noted numbness and tingling of both lower extremities. Not long afterward he had his first attack of carpopedal spasm.

Hospital studies * showed that this man's stools always contained from 75 to 80 per cent. of fat. He had persistent anaemia, and his blood chemistry was profoundly disturbed. At the onset of his disease, the serum calcium was 9.8 milligrams and the phosphorus 2.7 milligrams per 100 cubic centimeters. From then on the calcium was never higher than 8.2 milligrams and usually ranged between 5.4 and 6.8 milligrams. The corresponding phosphorus levels were 1.2 to 1.8 milligrams. His urinary excretion of calcium was very low, usually in the neighborhood of 10 milligrams per twenty-four hours. The phosphatase was never higher than 8.6 units (normal by the method used is 4.0).

A diagnosis of non-tropical sprue (idiopathic steatorrhoea) was made and the patient was treated with liver, calcium, vitamin D, and A.T. 10. His response was fair, but since he did not follow directions or report regularly, he had frequent acute relapses. These were often precipitated by minor intercurrent illnesses, such as colds.

In February 1943, twelve years after the initial symptoms, chest pain developed which was thought to be pleurisy. The pain grew worse and was accompanied by tenderness over several ribs on the right side. Roentgenograms showed osteoporosis and lesions in several ribs on both sides (Fig. 2-A) which were interpreted as pathological fractures. About a year after the onset of the chest pain, the patient began to complain that he "couldn't straighten up for a while after sitting down". He also had pain in the right tibia associated with weight-bearing. All of these symptoms improved after a short hospital stay, during which he had intensive therapy with calcium and vitamin D.

In September 1945, fourteen years after his disease began and two and one-half years after his first bone symptoms, the patient died rather suddenly of heart failure. It is interesting to speculate as to what role his deranged electrolytes may have played in precipitating cardiac failure. Two days before death his serum-potassium level was only 2.1 milliequivalents per liter. (On the same date the calcium was 5.7 milligrams per

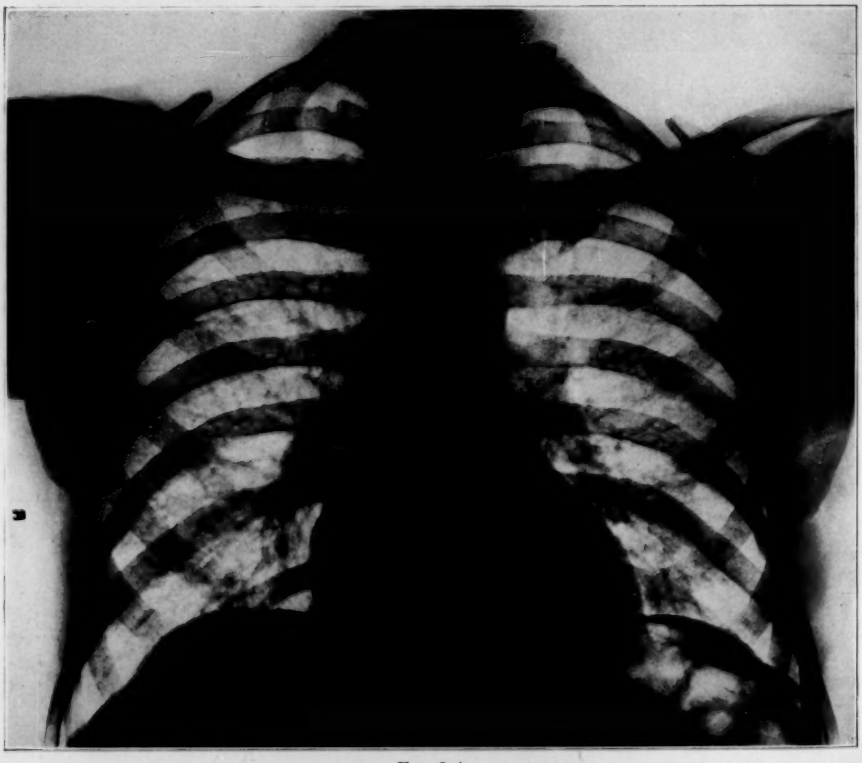

FIG. 2-A

Case 2. Roentgenogram of chest shows, in addition to generalized decrease in density, fracture of eighth rib on the left, and of seventh and eighth ribs on the right (in the axillary line).

* On the service of Dr. H. T. Ricketts.

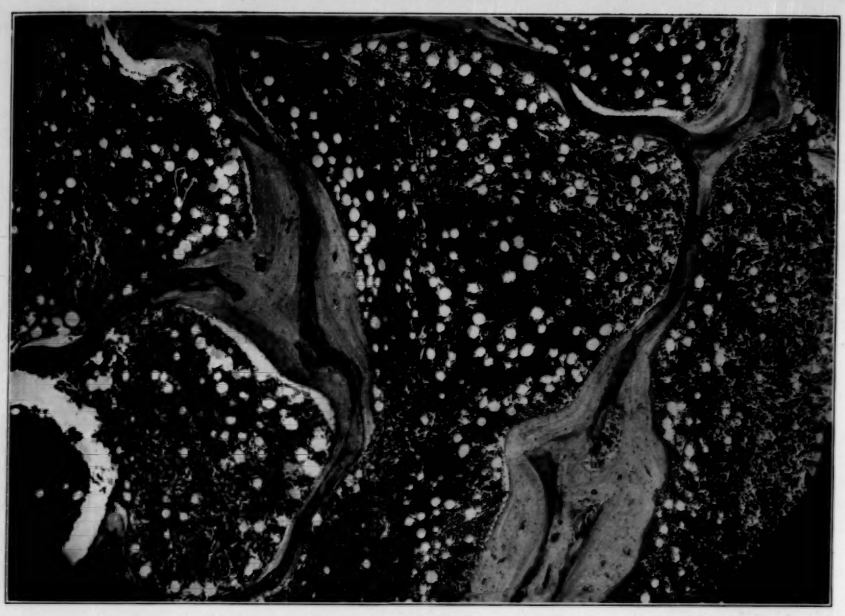

FIG. 2-B

Photomicrograph (× 40). (Compare with Fig. 1-D.) The darker-staining areas in the center of the trabeculae are bone. The remainder of the trabeculae are composed of osteoid tissue.

100 cubic centimeters; carbon dioxide 27.9 millimoles per liter; serum pH 7.44; and chlorides 97.6 millimoles per liter. The postmortem examination was unremarkable except for the gastro-intestinal tract and the bones. The parathyroids appeared normal to gross and microscopic examination.

Sections of the bones show the characteristic changes (Fig. 2-B). The trabeculae are normal in shape and distribution, but only a small portion of the center of each is calcified. The rest is osteoid tissue. The marrow is hyperplastic, but otherwise normal. There is no evidence of increased activity of the parathyroids.

COMMENT

The two patients described here are the fourth and fifth pathologically proved cases of osteomalacia to be reported from North America. The first such patient was the man reported by Elliott and Nadler and later by Gunn and Nadler; these authors worked before metabolic studies were generally available, but had excellent pathological material. In two of the cases reported by Albright and his associates the diagnosis was proved by microscopic examination.

Seven other cases, although not confirmed by pathological examination, seem unquestionable. These are four of Albright's patients, Milkman's patient, the patient of Miyakawa and Stearns, and the woman studied by Gargill, Gilligan, and Blumgart.

There are thus twelve acceptable cases. Of these, three were of unknown etiology, four followed steatorrhoea, three resulted from damage to the kidney tubules, and two depended upon a tendency of the kidneys to excrete too much calcium (idiopathic hypercalcuria).

In addition, there are six other reports (those by Bailey, Edeiken and Schneeberg, Eyermann, Tenney, Stone, and MacFarlane) which very likely concern osteomalacia, but which present insufficient data to prove that diagnosis.

It is obvious that osteomalacia is not so rare in North America as it has been believed to be. It is also obvious that it is by no means limited to females. Furthermore, since in this country simple dietary deficiency is a rare cause, osteomalacia most often appears as a complication of some other disorder. Therefore, if the possibility of bone changes be kept

in mind, more biopsies will be done and the diagnosis will probably be made with increasing frequency.

SUMMARY AND CONCLUSIONS

1. Osteomalacia (adult rickets) is a condition in which bone matrix (osteoid tissue) is formed normally, but fails to calcify.

2. Although osteomalacia from simple lack of vitamin D is rare in this country, the condition is not an uncommon complication of various gastro-intestinal and kidney disorders.

3. The diagnosis may be made with certainty by biopsy of bone, which will reveal abnormal amounts of osteoid tissue.

4. The term osteomalacia should be reserved to describe this condition and no other.

BIBLIOGRAPHY

1. Abbot: Mollities Ossium. Boston Med. and Surg. J., **93**: 72, 1875.
2. Albright, Fuller; Burnett, C. H.; Parson, William; Reifenstein, E. C., Jr.; and Roos, Albert: Osteomalacia and Late Rickets. The Various Etiologies Met in the United States with Emphasis on That Resulting from a Specific Form of Renal Acidosis, the Therapeutic Indications for Each Etiological Sub-Group, and the Relationship between Osteomalacia and Milkman's Syndrome. Medicine, **25**: 399–479, 1946.
3. Arcangeli: Osteomalacie, rachitisme et maladie osseuse de Paget. Arch. Gén. de Méd., **201**: 321–332, 1910.
4. Bab, Hans: Die Behandlung der Osteomalacie mit Hypophysenextrakt. Münchener Med. Wchnschr., **58**: 1814–1817, 1911.
5. Bailey, Harold: Report of a Case of Osteomalacia. Am. J. Obstet., **73**: 1076–1081, 1916.
6. Bartlett, Albert: Case of Mollities Ossium. Boston Med. and Surg. J., **22**: 205–206, 1840.
7. Bence-Jones, Henry: On a New Substance Occurring in the Urine of a Patient with Mollities Ossium. Philos. Trans., Royal Soc. London, Part I, pp. 55–62, 1848.
8. Blank, G. M., and Graves, G. W.: The Unusual Association of Postinfantile Rickets with Osteomalacia. Am. J. Dis. Child., **38**: 84–94, 1929.
9. Chu, H. I.; Liu, S. H.; Yu, T. F.; Hsu, H. C.; Cheng, T. Y.; and Chao, H. C.: Calcium and Phosphorus Metabolism in Osteomalacia. X. Further Studies on Vitamin D Action: Early Signs of Depletion and Effect of Minimal Doses. J. Clin. Investigation, **19**: 349–363, 1940.
10. Chu, H. I.; Yu, T. F.; Chang, K. P.; and Liu, W. T.: Calcium and Phosphorus Metabolism in Osteomalacia. VII. The Effect of Ultraviolet Irradiation from Mercury Vapor Quartz Lamp and Sunlight. Chinese Med. J., **55**: 93–124, 1939.
11. Cohnheim, Julius: Lectures on General Pathology. A Handbook for Practitioners and Students, Section 2. (Translated by A. B. McKee.) London, The New Syndenham Society, 1889.
12. Cristofoletti, R.: Zur Pathogenese der Osteomalazie. Gynäk. Rundschau, **5**: 113, 169–201, 1911.
13. Curschmann, Hans: Über den mono- und pluriglandulären Symptomenkomplex der nichtpuerperalen Osteomalacie. Deutsches Arch. f. Klin. Med., **129**: 93–117, 1919.
14. Dalton, N.: Mollites Ossium: Arrest and Cure of a Case of Mollites Ossium by the Use of Phosphate of Lime and Phosphoric Acid. Trans. Ohio State Med. Soc., **18**: 67–77, 1863.
15. Dalyell, E. J.; Sydney, Ch. M.; and Chick, Harriette: Hunger-Osteomalacia in Vienna, 1920. I. Its Relation to Diet. Lancet, **2**: 842–849, 1921.
16. Dwyer, H. L., and Eckelberry, O. S.: Osteomalacia in Children. A Study of the Mineral Metabolism. Am. J. Dis. Child., **31**: 639–653, 1926.
17. Edeiken, Louis, and Schneeberg, N. G.: Multiple Spontaneous Idiopathic Symmetrical Fractures. Milkman's Syndrome. J. Am. Med. Assn., **122**: 865–870, 1943.
18. Elliott, C. A., and Nadler, W. H.: The Effect of Castration upon Osteomalacia in the Male. Am. J. Med. Sciences, **153**: 722–732, 1917.
19. Erdheim, J.: Über Epithelkörperbefunde bei Osteomalacie. Sitzungsb. d. k. Akad. d. Wissensch. Math.-Naturw. Klasse, Wien, **116**: 311–370, 1907.
20. Eyermann, C. H.: Hunger-Osteomalacia. Report of a Case-Recovery. J. Missouri State Med. Assn., **29**: 551–554, 1932.
21. Fochier, A.: Sur les modifications récentes de l'opération césarienne, à propos d'un cas d'amputation utéro-ovarienne comme complément de cette opération. Lyon Méd., **31**: 393–399, 473–478, 505–510, 545–551, 1879.
22. Gargill, S. L.; Gilligan, D. R.; and Blumgart, H. L.: Metabolism and Treatment of Osteomalacia. Its Relation to Rickets. Arch. Int. Med., **45**: 879–907, 1930.

23. Goldthwait, J. E.; Painter, C. F.; Osgood, R. B.; and McCrudden, F. H.: A Study of the Metabolism in Osteomalacia. Am. J. Physiol., **14**: 389–402, 1905.
24. Gunn, F. D., and Nadler, W. H.: Osteomalacia. Necropsy Observations in a Man. Arch. Int. Med., **54**: 145–157, 1934.
25. Hadley, H. G.: Osteomalacia. Delaware State Med. J., **13**: 184–185, 1941.
26. Hannon, R. R.; Liu, S. H.; Chu, H. I.; Wang, S. H.; Chen, K. C.; and Chou, S. K.: Calcium and Phosphorus Metabolism in Osteomalacia. I. The Effect of Vitamin D and Its Apparent Duration. Chinese Med. J., **48**: 623–636, 1934.
27. Hess, A. F.: Rickets Including Osteomalacia and Tetany, p. 317. Philadelphia, Lea and Febiger, 1929.
28. Hoennicke, Ernst: Über das Wesen der Osteomalacie und seine therapeutischen Consequenzen. Ein Beitrag zur Lehre von den Krankheiten der Schilddrüse. Nebst Bemerkungen über den seelischen Zustand bei der Knochenerweichung. Samml. Zwangl. Abhandl. a. d. Geb. d. Nerven. u. Geisteskr., **5**: 1–78, 1905.
29. Holmes, G. W.: Osteomalacia. Its Roentgenographic Appearance and Classification. Am. J. Roentgenol., **5**: 507–512, 1918.
30. Hume, E. M., and Nirenstein, Edmund: Hunger-Osteomalacia in Vienna, 1920. II. Comparative Treatment of Cases of Hunger-Osteomalacia in Vienna, 1920, as Out-Patients with Cod-Liver Oil and Plant Oil. Lancet, **2**: 849–853, 1921.
31. Hutchison, H. S., and Stapleton, Grace: On Late Rickets and Osteomalacia. British J. Child. Dis., **21**: 18–33, 96–106, 1924.
32. Jampolis, Mark, and Londe, Sol: The Need of Larger Doses of Viosterol in Severe Rickets (Osteomalacia Type). A Comparative Study of the Efficacy of Viosterol and Cod Liver Oil. J. Am. Med. Assn., **98**: 1637–1640, 1932.
33. Jones, Joseph: Mollities Ossium. Trans. Am. Med. Assn.; **20**: 363–432, 1869.
34. Koch, Josef: Untersuchungen über die Lokalisation der Bakterien, das Verhalten des Knochenmarkes und die Veränderungen der Knochen, insbesondere der Epiphysen, bei Infektionskrankheiten. Mit Bermerkungen zur Theorie der Rachitis. Ztschr. f. Hyg. u. Infektionskr., **69**: 436–462, 1911.
35. Lamb, D. S.: Osteomalacia. J. Am. Med. Assn., **18**: 188–190, 1892.
36. Langendorff, O., und Mommsen, J.: Beiträge zur Kenntniss der Osteomalacie. Virchow's Arch. f. Path. Anat., **69**: 452–487, 1877.
37. Levacher de la Feutrie, A. F. T.: Traité du rakitis, ou l'art de redresser les enfants contrefaits. Paris, Lacombe, 1772.
38. Liu, S. H.: The Role of Vitamin D in the Calcium Metabolism in Osteomalacia. Chinese Med. J., **57**: 101–118, 1940.
39. Liu, S. H.; Ch'in, K. Y.; Chu, H. I.; and Pai, H. C.: Osteomalacia. Clinical, Metabolic, and Pathologic Studies of a Case with Parathyroid Hyperplasia and Right-Sided Cardiac Hypertrophy from Thoracic Deformities and Vitamin B_1 Deficiency. Chinese Med. J., **58**: 141–166, 1940.
40. Liu, S. H.; Chu, H. I.; Hsu, H. C.; Chao, H. C.; and Chen, S. H.: Calcium and Phosphorus Metabolism in Osteomalacia. XI. The Pathogenetic Rôle of Pregnancy and Relative Importance of Calcium and Vitamin D Supply. J. Clin. Investigation, **20**: 255–271, 1941.
41. Liu, S. H.; Chu, H. I.; Su, C. C.; Yu, T. F.; and Cheng, T. Y.: Calcium and Phosphorus Metabolism in Osteomalacia. IX. Metabolic Behavior of Infants Fed on Breast Milk from Mothers Showing Various States of Vitamin D Nutrition. J. Clin. Investigation, **19**: 327–347, 1940.
42. Liu, S. H.; Hannon, R. R.; Chu, H. I.; Chen, K. C.; Chou, S. K.; and Wang, S. H.: Calcium and Phosphorus Metabolism in Osteomalacia. II. Further Studies on the Response to Vitamin D of Patients with Osteomalacia. Chinese Med. J., **49**: 1–21, 1935.
43. Lobstein, J.-G.: Traité d'anatomie pathologique. Paris, F.-G. Levrault, 1833.
44. Looser: Über pathologische Formen von Infraktionen und Callusbildungen bei Rachitis und Osteomalakie und anderen Knochenerkrankungen. Zentralbl. f. Chir., **47**: 1470–1474, 1920.
45. MacFarlane, Foster: The Report of a Case of Osteomalacia, with Some Remarks. Maritime Med. News, **7**: 249–251, 1895.
46. McCrudden, F. H.: The Composition of Bone in Osteomalacia. Am. J. Physiol., **17**: 32–34, 1906.
47. McCrudden, F. H.: The Effect of Castration on the Metabolism in Osteomalacia. Am. J. Physiol., **17**: 211–217, 1906.
48. McCrudden, F. H.: Chemical Analysis of Bone from a Case of Human Adolescent Osteomalacia. J. Biol. Chem., **7**: 199–200, 1910.
49. McCrudden, F. H.: Studies of Bone Metabolism, Especially the Pathological Process, Etiology and Treatment of Osteomalacia. Arch. Int. Med., **5**: 596–630, 1910.
50. McCune, D. J.; Mason, H. H.; and Clarke, H. T.: Intractable Hypophosphatemic Rickets with Renal Glycosuria and Acidosis (The Fanconi Syndrome). Report of a Case in which Increased Urinary Organic Acids Were Detected and Identified, with a Review of the Literature. Am. J. Dis. Child., **65**: 81–146, 1943.

51. Mangum, C. P.: Osteomalacia—Report of Case. Southern Med. and Surg., **84**: 295–296, 1922.
52. Maxwell, J. P.: Osteomalacia in China. China Med. J., **37**: 625–642, 1923.
53. Miles, L. M., and Feng, Chih-Tung: Calcium and Phosphorus Metabolism in Osteomalacia. J. Exper. Med., **41**: 137–157, 1925.
54. Milkman, L. A.: Pseudofractures (Hunger Osteopathy, Late Rickets, Osteomalacia). Report of a Case. Am. J. Roentgenol., **24**: 29–37, 1930.
55. Milkman, L. A.: Multiple Spontaneous Idiopathic Symmetrical Fractures. Am. J. Roentgenol., **32**: 622–634, 1934.
56. Miller, B. F.: Report of a Case of Mollities Ossium, in Its Earlier Stage. Cincinnati Lancet and Observer, N. S., **13**: 117–121, 1870.
57. Miyakawa, George, and Stearns, Genevieve: Severe Osteoporosis (or Osteomalacia) Associated with Long-Continued Low-Grade Steatorrhoea. J. Bone and Joint Surg., **24**: 429–437, Apr. 1942.
58. Moers und Muck: Beiträge zur Kenntniss der Osteomalacie. Deutsches Arch. f. Klin. Med., **5**: 485–504, 1869.
59. Morhardt, P.-E.: Ostéomalacie fruste. Presse Méd., **48**: 479–480, 1940.
60. Naegeli: Uebersicht über die Symptomatik der Osteomalazie als innersekretorischer pluriglandulärer Erkrankung. Münchener Med. Wchnschr., **65**: 585–586, 1918.
61. Neill, John: Case of Mollities Ossium. Am. J. Med. Sciences, **68**: 119–126, 1874.
62. Olin, Harry: Osteodystrophy. Osteomalacia Osteopetrosis (Marble Bones). Report of Several Cases. Illinois Med. J., **75**: 517–521, 1939.
63. Pearson, J. E. G.: Idiopathic Steatorrhoea: Osteomalacia. Proc. Royal Soc. Med., **38**: 132, 1945.
64. Pommer, Gustav: Untersuchungen über Osteomalacie und Rachitis. Leipzig, F. C. W. Vogel, 1885.
65. Recklinghausen: Ueber osteomalacische Knochenstructuren. Centralbl. f. Allg. Path. u. Path. Anat., **8**: 858–859, 1897.
66. Russell, G. C.: Osteo-Malacia, with the Report of a Case. Cleveland Med. Gaz., **7**: 471–478, 1892.
67. Schmorl, G.: Die pathologische Anatomie der rachitischen Knochenerkrankung mit besonderer Berücksichtigung ihrer Histologie und Pathogenese. Ergeb. d. inn. Med. u. Kinderh., **4**: 403–454, 1909.
68. Schütte, D.: Beobachtungen über den Nutzen des Berger Leberthrans. Arch. f. Med. Erfahrung, **2**: 79–92, 1824.
69. Scipiades, Elemér: Erster durch Thymusimplantation geheilter Fall der Osteomalakie. Zentralbl. f. Gynäk., **48**: 1885–1888, 1924.
70. Scott, A. C.: A Contribution to the Study of Osteomalacia in India. Indian J. Med. Research, **4**: 140–168, 1916.
71. Short, D. W.: Osteomalacia Associated with Steatorrhoea: Report of a Case. Glasgow Med. J., **29**: 53–56, 1948.
72. Skinner, A. G.: Malacosteon. Boston Med. and Surg. J., **22**: 284–286, 1840.
73. Stone, L. R.: A Case of Osteo-Malacia with Autopsy. Boston Med. and Surg. J., **109**: 220–221, 1883.
74. Tenney, J. W.: Case of Mollities Ossium. Am. J. Med. Sciences, **26**: 506–508, 1839.
75. Trousseau, A., et Lasègue, Ch.: Du rachitisme et de l'ostéomalacie comparés. Union Méd., **4**: 314, 326–327, 354–355, 370–371, 385–386, 497–498, 514, 530–531, 537–538, 1850.
76. Virchow, Rud.: Das normale Knochenwachsthum und die rachitische Störung desselben. Virchow's Arch. f. Path. Anat., **5**: 409–507, 1853.
77. Weber, Otto: Zur Kenntniss der Osteomalacie, insbesondere der senilen und über das Vorkommen von Milchsäure in osteomalacischen Knochen. Virchow's Arch. f. Path. Anat., **38**: 1–15, 1867.
78. Wheaton, C. A.: Osteomalacia. Northwest. Lancet, **11**: 389–393, 1891.

TRANSPLANTATION OF THE EXTERNAL OBLIQUE MUSCLE FOR ABDUCTOR PARALYSIS *

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When the abductors of the hip are paralyzed, the weight of the body cannot be supported over the hip in the normal manner, and the paralytic, in order to balance his weight, must sway his body toward the affected side with each step. The swaying trunk gives these patients a distressingly awkward gait.

Many procedures have been described for the relief of this condition. Legg shifted the tensor fasciae latae backward on the ilium, establishing its line of pull to substitute for the glutaeus medius. Kreuscher stated that two procedures were introduced by Fritz Lange. One utilized a portion of the sacrospinalis, with silk-cord splicing, to substitute for the glutaeus medius and glutaeus minimus when the glutaeus maximus was intact; the other employed the vastus lateralis by means of a silk-cord attachment to the crest of the ilium. The use of fascia lata with silk-cord attachment to the trochanter was added by Kreuscher in an effort to diminish adhesions. Hey Groves combined the Legg and Lange operations by suturing the origin of the tensor fasciae latae to the sacrospinalis mass, and anchoring the tendon of the tensor fasciae latae at the base of the greater trochanter.

Telson substituted the anterior portion of the glutaeus maximus for the abductors of the thigh by transferring it forward on the ilium, as far as its blood and nerve supply would permit. Dickson transplanted the origin of the tensor fasciae latae to the region of the posterior superior spine of the ilium, to assist in abductor power. Mobilization of the iliotibial band was done by Ober. He drew the distal end of the band through a tunnel, just below the greater trochanter, and passed it upward under the gluteal fascia to attach it to a mobilized sacrospinalis mass. Mayer used a long tube of fascia attached to the lower end of the sacrospinalis; he implanted it into the posterior aspect of the femur for glutaeus maximus paralysis, and into the greater trochanter for glutaeus medius paralysis[7].

The disadvantages of long silk cords or strips of fascia lata for tendon splicing are obvious. The muscles recommended for transference have a limited range of contraction, and these procedures can be used only in the few patients who happen to have good power in the muscles to be transplanted. Steindler has expressed the feeling that none of these procedures is sound, and that appropriate joint fusion may be preferable.

The authors also had hoped that a method of eliminating the swaying gait might be found. However, we soon realized that, unless completely normal abductor power could be restored, the best that could be expected would be to assist the patient materially in maintaining balance upon a paralyzed extremity. This would lessen his fatigue and increase his endurance (Fig. 1).

Most of the muscles that have been suggested previously for transference receive their innervation from the same level of the spine as the glutaeus medius. The segments from the fourth lumbar to the first sacral, which innervate the abductors of the thigh, also innervate the tensor fasiae latae. The vastus lateralis is supplied by the segments from the second to the fourth lumbar, and the glutaeus maximus receives innervation from the fifth lumbar to the second sacral nerves. The sacrospinalis is supplied by the

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 27, 1949.

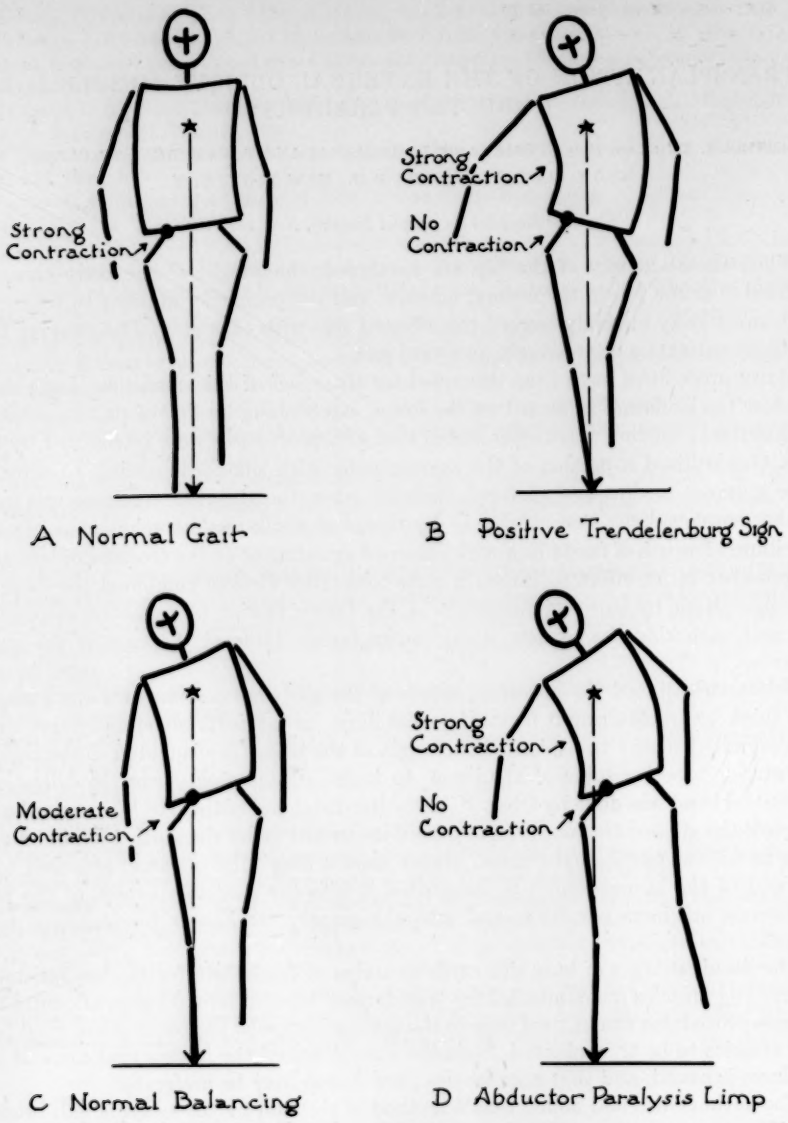

FIG. 1

Drawing to show variations in stance and gait.
A: Normal gait. *B:* Trendelenburg sign.
C: Normal balancing. *D:* Balancing when abductors are paralyzed.
Note: Muscle substitution can only reduce *D* to *C* and cannot restore *D* to *A*.

posterior primary divisions of the spinal nerves in the lumbar and lowest thoracic segments.

Above the hip region, however, a strong muscle, suitable for transplantation to the trochanter, often develops in the patient with abductor paralysis, particularly if that patient has been ambulatory for any length of time. The external oblique muscle of the abdomen becomes hypertrophied because it constantly assists in swaying the body back and forth with each step. It derives its nerve supply from the lower eight thoracic seg-

ments, and has a long, broad aponeurosis which can be transferred directly to the greater trochanter with no fascial splicing or silk-cord substitution. Its surfaces are adapted for gliding over the adjacent tissues, and its action on the trochanter can be established in a direct line from its broad origin on the thoracic cage. The muscles beneath it may also become hypertrophied; and, if the external oblique is strong enough to be suitable for transplantation, the other abdominal muscles will be adequate to maintain the integrity of the abdominal wall. The external oblique muscle of the abdomen, because of these characteristics, makes a good substitute for paralyzed abductors of the thigh.

Kreuscher stated: "Samter* and Krukenberg attempted to substitute the external oblique muscle for the gluteus medius and gluteus minimus." He does not indicate when or how this was done. Lowman described attaching the external oblique muscle to the greater trochanter by means of a strip of fascia lata, but he employed only a portion of the belly of the external oblique.

In 1939 one of the authors (L.I.T.) demonstrated anatomically that a segment of the external oblique muscle, with a piece of the external oblique aponeurosis acting as a tendon, could be transplanted to the greater trochanter. The first clinical trial of this procedure was made by another of the authors (T.C.T.) in August 1941. As no abdominal weakness was noted and as it seemed desirable to obtain as much abduction power as possible, the operation has subsequently been modified to include the entire external abdominal oblique muscle (Figs. 2-A and 2-B).

The purpose of this procedure is to provide active abductor power for the thigh when the glutaeus medius and glutaeus minimus are paralyzed. The external oblique muscle is completely freed from its normal insertion, and is mobilized up to its origin from the ribs. Its distal aponeurosis is inserted into the most prominent portion of the greater trochanter.

The skin incision is a long oblique one, passing from the pubic spine, over the crest of the ilium, to the costal margin in the posterior axillary line. Two incisions are made in the aponeurosis of the external oblique, about two centimeters apart, parallel to Poupart's ligament. These are carried down to within about one centimeter of the pubis, and joined. The superior incision is then directed upward along the medial border of the muscle belly of the external oblique to the costal margin. This frees the fibers of the external oblique from the remainder of its aponeurosis. The lower incision in the aponeurosis of the external oblique is extended laterally to the anterior superior spine of the ilium. The posterior border of the external oblique muscle is then identified at Petit's triangle. A blunt instrument is passed beneath the fibers of the external oblique, and the muscle fibers are cut across, close to their insertion into the iliac crest. The muscle belly is freed from the underlying structures by blunt dissection. The raw edge of the cut muscle is folded under and sutured onto itself, to form a cone-shaped muscle belly. The gap in the aponeurosis of the external oblique is closed from the pubis laterally, as far as possible.

Where the aponeurosis has been cut away from the muscle belly in the longitudinal direction, it is sutured down to the underlying internal oblique muscle. This leaves a small triangular defect in the outer layer of the anterolateral abdominal wall, but no weakness has been apparent clinically in any instance.

A lateral incision, six centimeters in length, is made over the most prominent part of the greater trochanter. The fascia and periosteum are incised in two places, and two quarter-inch drill holes are made in the most prominent part of the trochanter, at right angles to each other. A subcutaneous tunnel from this incision up to the original incision is formed, as far posteriorly as possible. This tunnel should be rather large where it crosses the crest of the ilium, to allow plenty of room for the external oblique muscle, which may form a mass five or six centimeters in diameter. The fascial tendon which has

* SAMTER, O.: Operativer Ersatz gelähmter Hüftmuskeln durch den Obliqu. externus. Zentralbl. f. Chir., **44**: 737–742, 1917.

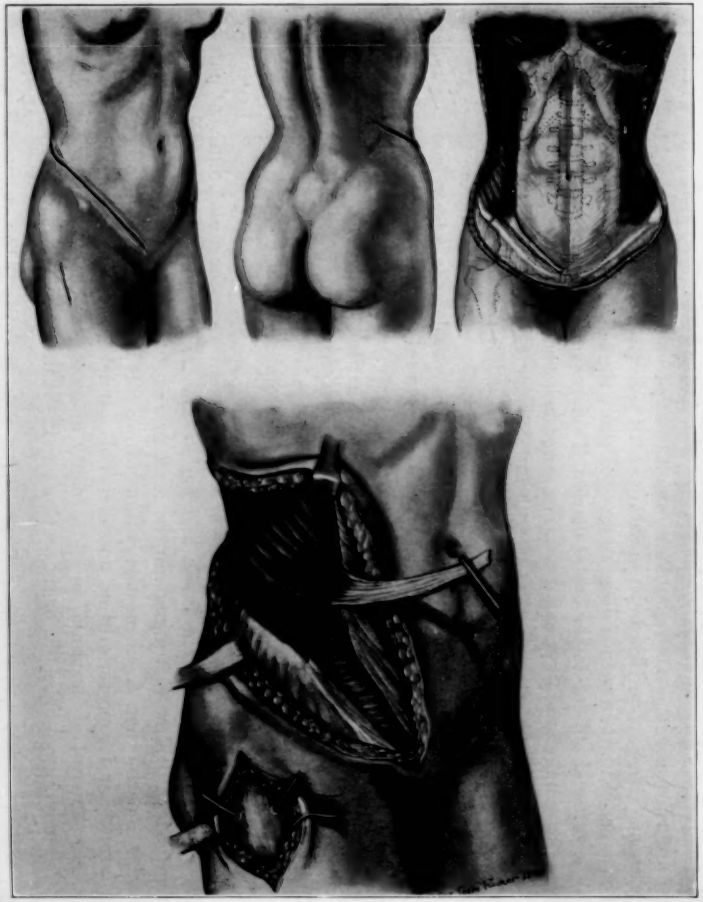

FIG. 2-A

FIG. 2-B

In operative procedure, the entire external oblique muscle is mobilized and transplanted.

TABLE I

RESULTS OF TRANSPLANTATION OF THE EXTERNAL OBLIQUE MUSCLE

FIG. 2-A

TABLE I

RESULTS OF TRANSPLANTATION OF THE EXTERNAL OBLIQUE MUSCLE

Case No. and Sex	Age at Onset of Poliomyelitis (*Years*)	Other Operations	Age at Operation (*Years*)	Status of Muscles of Affected Extremity	Shortening before Operation (*Cm.*)	Abduction Power	Strength of External Oblique	Date and Side of Operation	Results
1, F.	9	Astragelectomy, left	21	Poor. Generalized severe involvement of entire left extremity. Glutaeus maximus, fair; tensor fasciae latae, fair.	2.5	Poor	Normal	Aug. 22, 1941 Left	Good, 7 years. Transplant functions actively. Walking not limited by hip. Improvement in gait.
2, F.	1	Panarthrodesis, left foot; knee arthrodesis, left. Ober operation (for glutaeus maximus). Femoral shortening, right.	22	Poor. Left lower extremity almost flail. Hip flexors, poor; hip extensors, trace.	3	None	Good	Feb. 27, 1942 Left	Good, 2½ years. Transplant functions well horizontally; not against gravity. Lateral lurch decreased; stability improved subjectively.
3, F.	6	Foot stabilization, left.	13	Poor. Generalized severe involvement of entire left lower extremity. Hip flexors, good; hip extensors, fair; hip adductors, fair plus.	2	Trace	Fair plus	Mar. 4, 1942 Left	Good, 6½ years. Transplant functions actively against gravity. Limp only slightly improved; stability better. Patient very obese.
4, M.	2	Foot stabilization, left; tibial and fibular epiphyseodesis, right.	12½	Poor. Generalized severe involvement of left lower extremity. Hip flexors and extensors, fair plus.	1	Fair	Good plus	Apr. 1, 1942 Left	Excellent, 6½ years. Transplant rated at "good" against gravity and resistance. Patient walks 3 miles (5 blocks before operation). Limp reduced. Patient feels all improvement is due to hip operation.
5, F.	1	Opponens transplant, right; panarthrodesis, left foot.	22	Poor. Left leg flail. Hip flexors, fair; hip extensors, good.	5	Poor	Normal	Feb. 26, 1943 Left	Good, 6 years. Transplant functions against gravity only. Patient feels limp is less. Stability and endurance improved.

TABLE I (*Continued*)

Case No. and Sex	Age at Onset of Poliomyelitis (*Years*)	Other Operations	Age at Operation (*Years*)	Status of Muscles of Affected Extremity	Shortening before Operation (*Cm.*)	Abduction Power	Strength of External Oblique	Date and Side of Operation	Results
6, F.	2	Femoral epiphyseodesis, right. Peronaeus longus transplant, left; foot stabilization, left.	14	Poor. Extensive involvement of left lower extremity. Hip flexors, poor; extensors, good; adductors, poor.	0	Poor	Good minus	July 14, 1943 Left	Poor, 5½ years. Patient noticed no improvement. Transplant functions, but abduction against gravity is poor. Objectively, limp is slightly improved.
7, F.	3	Foot stabilization, left; lumbar sympathectomy, right; panarthrodesis, right foot; osteotomy, right tibia.	15	Very poor. Right lower extremity flail. Genu recurvatum. Hip flexors and adductors, nil; hip extensors, good.	2.5	None	Good	July 19, 1943 Right	Not rated, 5½ years. Transplant functions in horizontal plane, not against gravity. Because of painful recurvatum of knee, patient uses crutch; no useful weight-bearing.
8, M.	1	Femoral epiphyseodesis, left; upper tibial and fibular epiphyseodesis, left.	8	Poor. Entire right lower extremity practically flail. Hip flexors, good; hip extensors, fair; tensor fasciae, good.	6	None	Normal	Aug. 4, 1943 Right	Good
9, M.	3	Foot stabilization, left.	16	Fair plus. Left leg poor; thigh good. Hip flexors and extensors, normal.	2.5	Fair minus	Normal	Aug. 20, 1943 Left	Excellent, 5 years. Power of transplant rated at good plus. Patient able to partake in active sports; feels much improved.
10, F.	5	Foot stabilization, right; supracondylar osteotomy, right; tibial and fibular epiphyseodesis, left; transplant of peronaeus longus, right; ankle arthrodesis, right.	13	Poor. Right leg severely involved. Tensor fasciae, fair plus; hip extensors and adductors, good.	4.5	Poor	Normal	Oct. 20, 1943 Right *	Good, 5 years. Transplant functions against gravity and resistance. Limp slightly improved; stability greatly improved.

onaeus longus, right; ankle arthrodesis, right.

greatly improved.

11, F.	13	Bilateral astragalectomy; tenodesis, left wrist. Biceps femoris transplant, right; panarthrodesis, right ankle.	34	Very poor. Extensive involvement, upper and lower extremities. Right hip flexors, fair; hip adductors, poor; hip extensors, fair plus.		Poor	Good minus	Feb. 21, 1944 Right *	Fair, 4½ years. Definite improvement. Transplant can be felt to contract. Complicated by generalized involvement; crutch-walking essential.
12, F.	1	None	9	Fair. Generalized but not complete paralysis in right thigh and calf. Hip flexors, extensors, and adductors, good.	2	None	Normal	Apr. 5, 1944 Right	Fair (?), 4 years. Transplant contracts actively against gravity; abduction through only 10 degrees. Limp only slightly improved. (Too small a muscle segment utilized?)
13, F.	1	Posterior bone block, left ankle; ankle arthrodesis, right.	16	Poor. Extensive involvement of entire right lower extremity. Hip flexors and extensors, good.	Equal	None	Good	June 14, 1946 Right	Poor, 2 years. Transplant functions in horizontal position, but not against gravity. Patient uncooperative; reports no improvement.
14, M.	2	Foot stabilization, left. Epiphyseodesis (femoral, tibial, and fibular), right. Soutter operation, Ober operation, left.	18	Poor. Involvement of entire left lower extremity. Hip flexors and extensors, fair.	1.5 cm. overgrowth	Poor	Good	Nov. 8, 1946 Left	Not rated. No follow-up.
15, F.	5	Interphalangeal arthrodesis, left first toe. Foot stabilization, left. Femoral shortening, right.	17	Poor. Generalized involvement of left lower extremity. Hip flexors and extensors, good.	Equal	Trace plus	Normal	July 16, 1947 Left *	Excellent, 1½ years. Abducts well against gravity. Limp and stability much improved.
16, M.	5	Bilateral stabilization of foot. (Same admission as for oblique transplantation.)	35	Very poor. Extensive involvement of both lower extremities below hips. Hip flexors and extensors, good (left).	1.5	None	Normal	July 25, 1947 Left †	Good, 1½ years. Transplant functions very strongly. Progress slow because of concomitant foot operations, but improvement.

TABLE I (*Continued*)

Case No. and Sex	Age at Onset of Poliomyelitis (*Years*)	Other Operations	Age at Operation (*Years*)	Status of Muscles of Affected Extremity	Shortening before Operation (*Cm.*)	Abduction Power	Strength of External Oblique	Date and Side of Operation	Results
17, F.	1	Osteotomy of femur, right. Panarthrodesis, right ankle.	31	Very poor. Flail below right knee; poor above. Right hip flexors, extensors, and adductors, good.		Poor	Normal	Sept. 20, 1947 Right †	Fair, 1 year. Transplant functions well. Abducts in horizontal plane, but not against gravity.
18, F.	2½	Astragalectomy, left. Foot stabilization, right.	17	Very poor. Severe involvement, both lower extremities. Recurvatum, both knees. Right hip flexors and extensors, good.	2 cm. longer on right	Poor	Good plus	Sept. 24, 1947 Right †	Fair, 1 year. Transplant functions in horizontal position, but not against gravity. Stability improved; limp remains because of severe generalized involvement.
19, M.	3	Ankle arthrodesis, right.	14	Fair. Flail below right knee, but thigh is fair. Right hip flexors, good plus; hip extensors, good.	4	Trace	Normal	Feb. 4, 1948 Right †	Good, 10 months. Good power in transplant; abducts against gravity. Patient feels limp is less and gait is more stable. Improving.
20, M.	2	Astragalectomy, right. Femoral, tibial, and fibular epiphyseodesis, left. Revision of astragalectomy, right.	15	Poor. Practically flail right lower extremity. Other limbs good. Right hip flexors, nil; hip extensors, fair plus.	Equal	Fair	Normal	Apr. 7, 1948 Right * †	Not rated—too early. Transplant functions very well. Improvement slowed by concomitant foot procedure.
21-A, F.	2	Bilateral stabilization of foot. Spine arthrodesis, ninth thoracic to fourth lumbar.	19	Poor. Left leg poor; thigh fair. Left hip flexors and extensors, good.	1 cm. longer on left	Poor	Normal	May 28, 1948 Left †	Not rated—too early. At 6 months, left hip can be abducted against gravity. Improved stability; limp persists.

21-B				Very poor. Flail extremity with long leg brace. Right hip flexors, poor; extensors, trace plus.	1	Poor	Normal	June 11, 1948 Right †	Not rated.
22	6 mos.	Foot stabilization, right. Biceps femoris transplant, right. Femoral shortening, right; ankle arthrodesis, right.	12	Very poor. Left lower extremity flail; brace used. Right poor. Left hip flexors and extensors, trace.	1	Trace	Fair plus	July 21, 1948 Left †	Not rated—too early. At 6 months, abducts against gravity. Gait and stability improved. Progress slow because of concomitant procedures.
23, F.	9 mos.	Astragalectomy, right. Colonna capsuloplasty, right, for dislocated hip, Oct. 18, 1948.	32	Poor. Right foot fixed. Knee fair. Right hip flexors fair plus; right hip extensors poor plus.	3.5	None	Normal	Nov. 6, 1948 Right †	Not rated—too early.
24-A‡, M.	22	Nerve graft, cauda equina, Feb. 1944. Bilateral Ober operation (gluteus maximus), Aug. 10, 1944.	23	Poor. Both lower extremities flail. Quadriceps, good. Hip flexors and adductors, good; hip extensors, nil.	Equal	None	Normal	June 10, 1944 Right †	Fair. Bilateral transplants for paraplegia. Ober operations failed, but abduction is well done in horizontal plane, though not against gravity. Complicated by multiple fractures in 1945.
24-B‡						None	Normal	June 24, 1944 Left †	Fair.
25, F.	19	None	24	Poor. Flail below knee. Hip flexors, adductors, and extensors, poor.	Equal	None Tensor fasciae latae fair	Normal	Jan. 15, 1949 †	Not rated—too early.

* Tensor fasciae latae used. † Entire oblique muscle used. ‡ Bilateral transplants. Gunshot wound at third lumbar with paraplegia, Aug. 2, 1943.

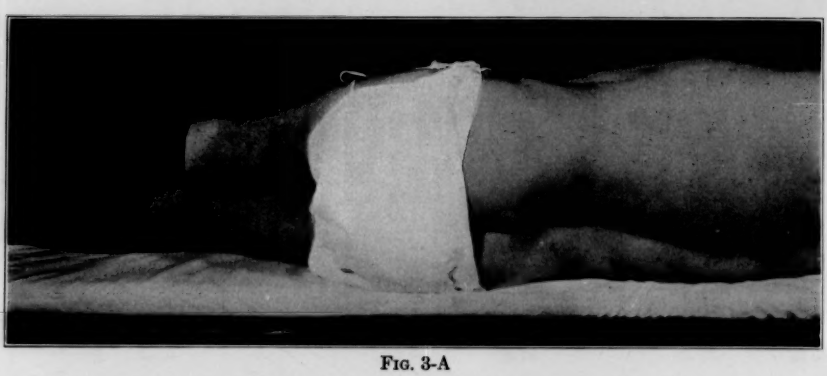

FIG. 3-A

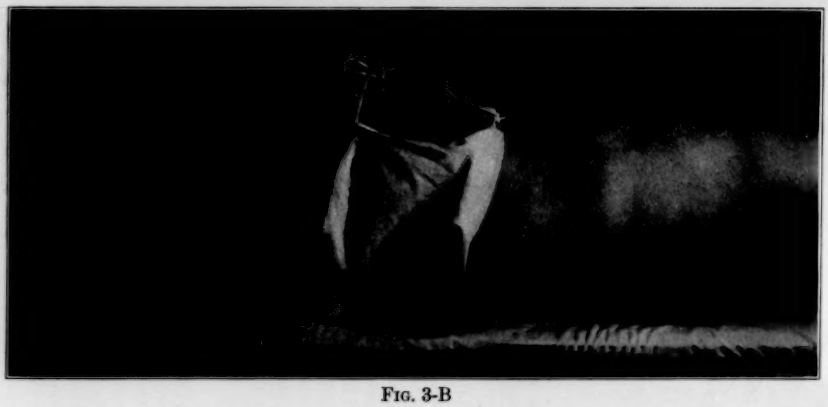

FIG. 3-B

Transplanted external oblique muscle, acting directly upon the trochanter, produces active abduction.

been made from the aponeurosis of the external oblique is passed downward through this tunnel. The muscle fibers then run in a longitudinal direction, from the lower rib margins over the crest of the ilium, directly toward the greater trochanter. The thigh is brought into wide abduction, and the tendon of the transplanted external oblique muscle is passed through the drill holes in the greater trochanter and sutured upon itself.

After wound closure, a plaster spica is applied with the hip in wide abduction. This spica remains in place for four weeks, and then abduction exercises are begun. The patient is given pool therapy to encourage abduction, but full weight-bearing is not permitted until eight weeks after the operation.

For this operation to be successful in stabilizing the hip during weight-bearing and in producing active abduction, the external oblique muscle must be strong. If the tensor fasciae latae is active, this may also be used to reinforce the abductor power. If the glutaeus maximus is strong, the superior part of this muscle may be moved forward to assist in active abduction. If the glutaeus maximus is paralyzed, extension of the hip may be obtained by combining transplantation of the external oblique with the Ober operation, the tensor fasciae latae and the sacrospinalis being used as substitutes for the glutaeus maximus.

Twenty-seven operations have been performed upon twenty-five patients (Table I). In two instances the operation was done on both sides. Twenty patients had had poliomyelitis before the age of five. Three had had poliomyelitis at the ages of six, nine, and thirteen, respectively. One patient was a soldier who had paraplegia, due to a gunshot

wound at the level of the third lumbar vertebra. The age at operation ranged from eight to thirty-five years, with an average age of nineteen. The average period from onset of poliomyelitis to transplantation of the external oblique was fifteen years.

RESULTS

One result was not rated, as the patient had to continue with crutches because of genu recurvatum; one patient did not return for follow-up examination; and six operations have been done too recently to be evaluated. It has been difficult to judge accurately the result of the transplantation, because generalized paralytic involvement has usually been present. The abductor limp has not been completely eliminated in any instance, but all patients have shown some improvement in abductor power.

The results of nineteen operations have been graded as follows: Three were considered as having excellent results, because of marked subjective and objective improvement. It should, however, be mentioned that each of these patients had slight abductor power before operation. Eight results were rated as good, because there was sufficient power in the transplant to permit abduction against gravity (Figs. 3-A and 3-B). In the six transplants rated as fair, the improvement in gait and stability was less, but still sufficient to justify the procedure. Even in the two patients whose results were graded as poor, some contraction could be felt in the transplant, but the patients reported no improvement.

CONCLUSIONS

1. The abductor limp is not eliminated by transplantation of the external oblique muscle, but it may be lessened.

2. Most patients have reported a substantial increase in their sense of security and an improvement in their gait.

3. Transfer of the insertion of the external oblique muscle to the greater trochanter has reduced fatigue and increased endurance.

REFERENCES

1. Dickson F. D.: An Operation for Stabilizing Paralytic Hips. A Preliminary Report. J. Bone and Joint Surg., **9**: 1–7, Jan. 1927.
2. Groves, E. W. Hey: Some Contributions to the Reconstructive Surgery of the Hip. British J. Surg., **14**: 487–517, 1927.
3. Kreuscher, P. H.: The Substitution of the Erector Spinae for Paralyzed Gluteal Muscles. An Operation for Stabilizing the Hip. Surg., Gynec., and Obstet., **40**: 593–597, 1925.
4. Legg, A. T.: Tensor Fasciae Femoris Transplantation in Cases of Weakened Gluteus Medius. New England J. Med., **209**: 61–62, 1933.
5. Lowman, C. L.: Lateral Fascial Transplant for Controlling a Gluteus Medius Limp. Physiotherapy Rev., **27**: 355–357, 1947.
6. Mayer, Leo: The Physiological Method of Tendon Transplantation. I. Historical; Anatomy and Physiology of Tendons. Surg., Gynec., and Obstet., **22**: 182–197; 298–306; 472–481, 1916.
7. Mayer, Leo: Personal communication.
8. Ober, F. R.: An Operation for the Relief of Paralysis of the Gluteus Maximus Muscle. J. Am. Med. Assn., **88**: 1063–1064, 1927.
9. Steindler, Arthur: Orthopedic Operations. Indications, Technique and End Results. Springfield, Illinois, Charles C. Thomas, 1940.
10. Telson, D. R.: Transplantation of the Gluteus Maximus for Paralyzed Gluteus Medius. Surg., Gynec., and Obstet., **46**: 417–419, 1928.

DISCUSSION

Dr. Frank D. Dickson, Kansas City, Missouri: That so many methods have been devised to recapture abduction power in paralyzed hips, for the purpose of gaining additional stability and overcoming the resultant swaying gait, indicates that none has been altogether successful. The authors of this paper have apparently recognized this situation, and they have added a new operation to our armamentarium.

Basically, in order to improve stability in a partially paralyzed hip, not only must abduction power be recaptured or reinforced, but also extension power must be at least adequate. In other words, it is my opinion, based upon clinical observation, that stability in the hip joint results from the coordinated action of a num-

(*Continued on page 225*)

PRIMARY TENORRHAPHY OF THE FLEXOR TENDONS IN THE HAND*

BY VINTON E. SILER, M.D., CINCINNATI, OHIO

From the Department of Surgery, College of Medicine, University of Cincinnati, and the Hand Clinic, Cincinnati General Hospital

The seriousness of wounds of violence in the hand and forearm has been stressed by Garlock, Koch and Mason [7], Mason[9], Bunnell, O'Shea, Winfield, Miller, and others. Some of these observers have stressed the fundamental principles which must guide every surgeon entrusted with the surgical repair of such injuries. Restoration of function occurs in direct ratio to the surgeon's ability to control all the factors which influence wound healing. The management of injuries of the hand, as of no other region of the body, presents a challenge in the application of these principles.

The purpose of this paper is to stress the fundamental principles of emergency, operative, postoperative, and follow-up treatment, and to report the results in eighty-four cases of severed flexor tendons in the hand, treated in the Cincinnati General Hospital fom 1941 to 1946, inclusive.

TREATMENT

Emergency Treatment

Emergency treatment for wounds on the volar aspect of the hand is the same as that for other wounds of violence. Good treatment consists in control of hemorrhage, treatment of shock, avoidance of further contamination, and, when indicated, chemotherapy and antitetanus therapy. In so far as possible, a correct diagnosis should be made by careful local and general physical examination.

Hemorrhage in the hand is usually controlled easily by means of a well applied pressure dressing or by compression of the major arteries at the wrist, if one cares to observe the wound more closely. The use of a tourniquet is not indicated unless amputation is imperative. After inspection of the wound, the pressure dressing should remain undisturbed until definitive therapy has been instituted. Such wounds should not be probed, and the blood vessels should not be clamped and ligated haphazardly during the period of emergency care. If there is evidence of hemorrhagic shock, therapy should be instituted quickly.

Every attempt should be made to lessen further contamination of the wound. In our Clinic we advocate that those in immediate contact with open wounds cover the nose and mouth with a face mask and, above all, that the surgeon cleanse his hands thoroughly before beginning a systematic examination of the injured hand. Details of this principle have been stressed by many observers, including Mason[10, 11], Koch[5, 6], Altemeier[1], and Siler.

In most instances intramuscular penicillin may be started during this period. Antitetanus therapy should always be given.

One of the most important phases of emergency treatment is a careful local physical examination. Only a systematic examination of the various muscles of the hand and forearm with regard to their specific functions will determine the presence or absence of injury to a tendon. Furthermore, a careful neurological examination on the basis of anatomical knowledge is the only method by which injury to nerves can be determined. In the majority of cases, one should be able to diagnose accurately before operation the vital structures damaged and to foretell operative expectations.

* Read at the Annual Meeting of the American Society for Surgery of the Hand, Chicago, Illinois, January 21, 1949.

Operative Treatment

In doing primary tenorrhaphy the author has, since 1940, followed the principles advocated by Mason[12]. There can be no doubt but that tendon repair demands operating-room conditions, small instruments to do delicate surgery, and the willingness and patience to see that all damaged structures are properly managed so as to afford the best functional results. Likewise, general anaesthesia is preferred unless there is a contra-indication, such as a condition of extreme intoxication. In 1941, an intoxicated patient died on the operating table at the completion of a long procedure, involving primary tenorrhaphy of the flexor tendons of the hand. Under such circumstances, we have used regional nerve block (brachial plexus) with novocain to the best advantage; or, more frequently, we have resorted to secondary tenorrhaphy after simple closure of the initial wound.

The use of the blood-pressure cuff, applied on the arm as a tourniquet at a pressure of 260 millimeters of mercury, after drainage of all venous blood for five minutes, ensures an avascular field and permits visibility afforded by no other method. We have seen no bad effects from this procedure.

For skin preparation, white bar soap and warm water have been used, or ceepryn chloride in a strength of 1 to 100. Cleansing is done after shaving the forearm and hand, and is not followed by the application of antiseptics. Excess cleansing agents are removed by irrigation with physiological saline solution. Viable tissue is not removed from the hand for, as Mason has said, there is no tissue to spare. Only devitalized skin, subcutaneous tissue, fat, and muscle are removed by débridement. Any dirt or foreign body is removed. All instruments used for this portion of the procedure are regarded as contaminated and are discarded. After the operative team has changed gowns and gloves (the instrument nurses having remained clean), one may begin the reconstructive phase of the operation.

Silk sutures have been used in all of the cases reported here; 6–0 silk is used to ligate small vessels and 4–0 silk for larger ones. Tenorrhaphy has been accomplished with silk of the 3–0 size.

Our policy has been to repair immediately all damaged flexor tendons in the palm of the hand, except those in the vicinity of the metacarpophalangeal joint, where we are convinced that to suture the flexor profundus alone or the proximal flexor sublimis to the distal flexor profundus affords a much better functional result than to attempt anatomical approximation of both. In the fingers we are sure this principle is sound, and here the ulnar and radial insertions of the flexor sublimis tendon should be carefully dissected away from their attachment to the second phalanx. The proximal end is also resected. This reduces tendon bulk, thereby allowing for freedom of motion of the remaining tendon. We believe that more skill is required in the repair of the flexors of the thumb than for other tendons in the hand. Divided nerves (branches of the median and ulnar nerves) require primary neurorrhaphy, which, except in badly mangled wounds, can usually be accomplished. The distal divided structures are usually identified first since they retract less; after they have been identified, one has a distinct advantage in locating the proximal ends. Counter incisions should be used to isolate the proximal ends of divided tendons. By using traction sutures of silk, one can replace the tendon in its bed. Cutting a window in the tough fibrous tendon sheath has been done in only a few cases, where the line of repair of the flexor profundus lay directly over the metacarpophalangeal joint.

The method of tendon suture used has been predominantly that advocated by Mason and Allen[13], and is shown graphically by their illustration (Fig. 1). Less frequently, Bunnell's method has been applied, and on a few occasions the Lange suture has been used. When laceration of the tendon only has occurred, interrupted sutures of a fine silk have been placed to make the correct approximation of this injury.

In all cases of primary tenorrhaphy, we close the wound, even using a skin graft if needed. This principle is postulated on the basis of control of hemorrhage. The pneumatic cuff is released immediately after repair of the tendon, and all bleeding points are clamped

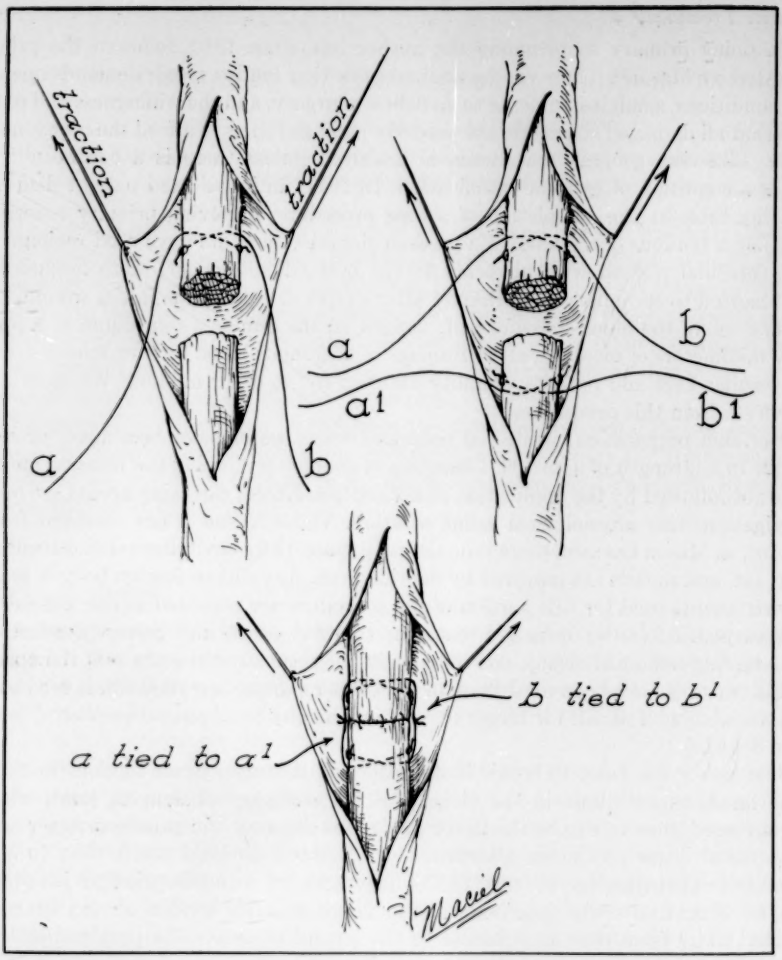

FIG. 1

Illustration of the Koch-Mason-Allen type of tendon repair. (Redrawn by permission of *J. B. Lippincott Company* from Fig. 1 *in* The Rate of Healing of Tendons by M. L. Mason and H. S. Allen. Ann. Surg. **113,** 428, 1941).

and ligated. The edges of the skin should close without tension. A mild pressure dressing is applied with the hand and forearm placed on a dorsal aluminum splint, molded and padded to fit the patient, which holds the digits in flexion, but separated from each other by flat gauze sponges.

Postoperative Treatment

Very little care is needed in the period immediately after operation. The forearm and hand are kept elevated to an optimum position in regard to the vascular system. The dressing should be checked several times daily during the first three days after operation for unusual discomfort of the fingers, position of the splint, and appearance of the skin of the arm. Usually these patients are required to walk, the forearm dressing being supported in a sling.

Although in the series of cases reported here, antibiotics were not used routinely, we

now administer 300,000 units of intramuscular penicillin for six days after operation. In those cases where there is great damage to the hand or associated injury to the body, the dosage may be increased. In some cases sulfadiazine and streptomycin are also used. On the basis of observation by Altemeier[2], we do not use gas-gangrene antitoxin.

The postoperative course decides the hospital discharge date. Usually the patient is sent home by the seventh or eighth day, after which he is followed in the Hand Clinic. Unless there is good reason, the initial dressing is not disturbed until the twelfth day, at which time the sutures are removed and active motion (in flexion) is begun. The splint is adjusted and is reapplied for another week. By the end of twenty-one days after primary tenorrhaphy the splint is used to support the hand, but is arranged for removal by the patient so that daily hydrotherapy (soaking the forearm and hand in a soapy bath) can be done at home. The splint is usually discontinued by the twenty-eighth day. Koch and Mason[8] have referred to this principle as "purposeful splinting".

Follow-up Treatment

Follow-up care is very important to the patient who has sustained tendon injury. Repeated observations show the progress being made and afford the only control of physical therapy. Furthermore, these patients become discouraged easily and can be helped tremendously by kindly advice. It becomes obvious that such a routine can be planned only in a central hand clinic, where attention can be given to details and complications can be wisely managed.

This is the period when the patient needs careful instruction regarding passive joint motion, and exercises which he can do without the physical therapist. Squeezing a soft sponge-rubber ball, both when in the arm bath and out of it, is an excellent exercise to promote function. The degree of disability can be properly evaluated only by careful follow-up examinations over a period of several months.

ANALYSIS OF CLINICAL MATERIAL

Almost without exception, all of those concerned with tenorrhaphy agree that the flexor tendons on the volar surfaces of the fingers present a difficult problem of repair.

TABLE I

ANATOMICAL DISTRIBUTION OF INJURY

Location of Injury	No. of Cases	Per cent.
Thumb	13	16
Finger	40	48
Palm	30	36
Total	83	100

The author's clinical observations have emphasized this point. Similarly, we know that much better results are generally obtained by primary tenorrhaphy to the extensor tendons in the hand and to the flexor and extensor tendons of the wrist. The purpose of this analysis is to demonstrate several factors which have influenced our results in the treatment of eighty-four cases of primary tenorrhaphy of the flexor tendons in the hand.

Incidence

From 1941 to 1946, inclusive, a total of 184 tendon injuries of the forearm and hand were treated at the Cincinnati General Hospital, so that the series studied represents about 46 per cent. of the total number of cases. The average age of the patients was

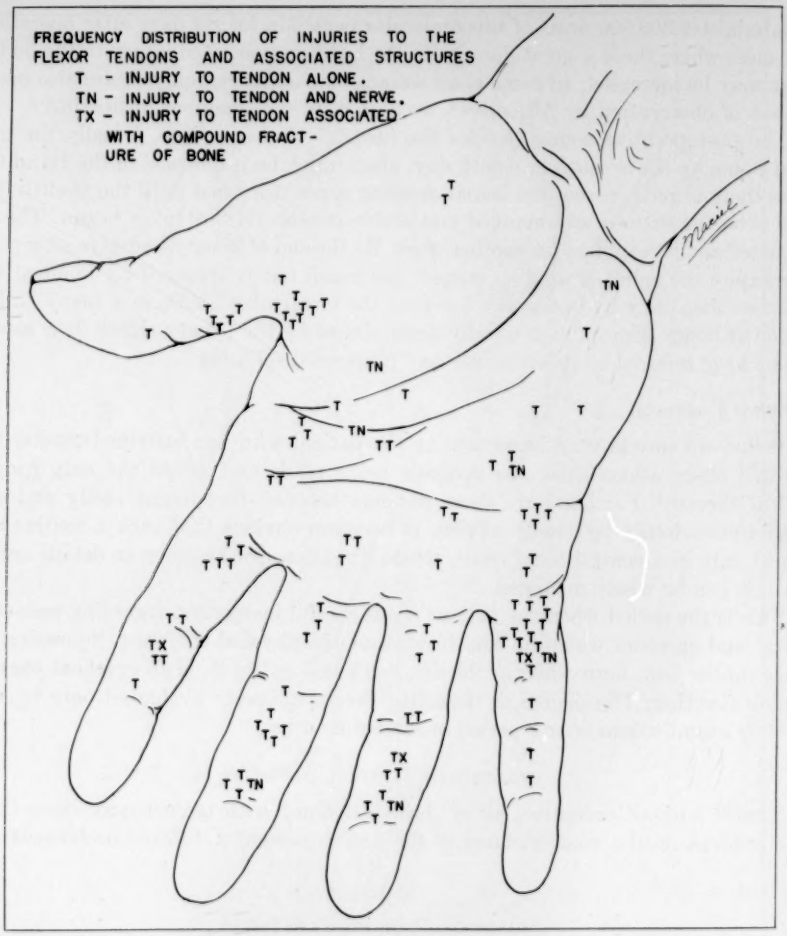
FREQUENCY DISTRIBUTION OF INJURIES TO THE FLEXOR TENDONS AND ASSOCIATED STRUCTURES
T - INJURY TO TENDON ALONE.
TN - INJURY TO TENDON AND NERVE.
TX - INJURY TO TENDON ASSOCIATED WITH COMPOUND FRACTURE OF BONE

FIG. 2

Diagrammatic view of the volar aspect of the hand, showing the frequency distribution of injuries to the flexor tendons and associated structures.

twenty-seven years. The wound of violence was produced by a knife in 52 per cent. of the cases; in the remainder the causative agent was glass, a saw, porcelain, metal strips, a grinder, a razor, an electric fan, a cleaver, or a hammer.

Of the 83 patients * available for study, thirty-four (41 per cent.) were white and forty-nine (59 per cent.) were negro. The injury is seen predominantly in the male, there being sixty-one males (73 per cent.) and twenty-two females (27 per cent.) in the series.

Location of Injury

The majority of wounds on the volar aspect of the hand occurred in the fingers, rather than in the palm (Fig. 2). If, for anatomical reasons, the location is analyzed further, the flexor tendons of the thumb were injured in thirteen (16 per cent.) of the cases, the fingers in forty (48 per cent.), and the palm in thirty (36 per cent.). The total number of injuries to digits was fifty-three (64 per cent.) as compared to thirty (36 per cent.) in the palm of the hand (Table I).

* One patient died on the operating table in 1941, giving a mortality rate for this series of 1.2 per cent.

TABLE II

DISTRIBUTION OF THE VARIOUS METHODS OF TENDON SUTURE

Method	No. of Cases	Per cent.
Koch-Mason-Allen	60	72
Bunnell	10	12
Lange	2	2
Combined	3	4
Other	8	10
Total	83	100

Time Factor

The best results are obtained when definitive treatment is begun soon after the injury. In wounds of violence, the longer the interval before treatment, the greater the incidence of infection. Wounds on the volar aspect of the hand, involving the flexor tendons, should be treated as soon as possible. Mason[12] believes that the best results are obtained if treatment is carried out within two hours after injury, while other observers extend the limit to three hours. The lapse of six hours or longer precludes the possibility of primary tenorrhaphy; and we agree with Koch, Mason, and Allen that simple cleansing of the wound with closure of the skin should be done, rather than tendon repair. In this series of cases, the average time between injury and the institution of definitive treatment was 3.8 hours. Depending upon the course of the wound, secondary tenorrhaphy can be done later. In such cases the use of chemotherapy may alter the time of repair.

Associated Injury

Damage to all nerves and bone fractures were included in this category: In sixty-four (77 per cent.) of the cases, there was no associated injury, whereas in nineteen (23 per cent.) associated injury was present. In each case an effort was made to manage the associated injury so as to obtain a good functional result.

Suture Material and Method of Tendon Suture

The Koch-Mason-Allen modification of the Max Lange suture has been used as the method of choice in tendon repair (Table II). This technique was applied in sixty (72 per cent.) of the cases. Mason has pointed out the advantages of this suture, and the

TABLE III

ANALYSIS OF RESULTS IN SIXTY-FIVE CASES AT FOLLOW-UP EXAMINATION

Location	No. of Cases	Results							
		Excellent		Good		Fair		Poor	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Thumb	9	3	33	5	55	1	11		
Finger	32	3	9	17	53	9	28	3	9
Palm	24	9	37	11	46	1	4	3	12
Total	65	15	23	33	51	11	17	6	9

experimental work of Mason and Allen presents supporting evidence of its efficiency. On fewer occasions, particularly in children, the Bunnell suture was applied. The Lange suture was used occasionally, and in some instances a combination of these methods was instituted. In those cases where partial division from laceration had occurred, associated with division of other tendons in the same hand, the area of partial division was repaired with interrupted sutures only. In those cases, with division of the flexor profundus at its insertion on the base of the distal phalanx, various modifications of methods were used.

Chemotherapy and Wound Infection

During the period from 1941 to 1946, there was no unanimity in regard to the exact status of chemotherapy. Furthermore it was the war period, when chemotherapeutic agents were difficult to obtain. Less than half (45 per cent.) of these patients with injury of the flexor tendons received chemotherapy, and yet only seven (8 per cent.) had infected wounds. In only three cases treated in 1943, was any chemotherapeutic agent placed in these wounds.

RESULTS

The results of this survey are not striking, but they certainly are not entirely different from those reported by other observers. The criteria by which the results were tabulated are as follows: An *excellent* result implies 80 per cent. or more of normal function without damage to nerves. A *good* result includes all cases with 50 per cent. or more of normal function without nerve impairment. A *fair* result includes those cases with less than 50 per cent. of normal function, with or without nerve involvement. A *poor* result refers to those cases with less than 30 per cent. of normal function, associated nerve damage, and usually ankylosis of the joints.

Excellent results were not common (Table III). They occurred most frequently in the palm of the hand (37 per cent.), next in the thumb (33 per cent.), and last in the finger (9 per cent.). Good results were more frequent and were obtained in about equal proportions in the various locations. If we combine the excellent and good results into a single *satisfactory* group, we have a total of 48 cases or 74 per cent. By combining the fair and poor results into an *unsatisfactory* group, we have a total of 17 cases, or 26 per cent. Eighteen cases or 22 per cent. had incomplete follow-up examinations. This is explained by the large percentage of negroes in the total series, and the fact that our hospital cares for many people on a transient basis. We have no means of following these patients.

A final observation concerns the number of surgeons doing primary tenorrhaphy of flexor tendons in the hand. During this five-year period, a total of thirty-five surgeons in the Graduate School of Surgery repaired the tendons in the eighty-three cases reported here. One assistant resident operated upon seven patients (the greatest number); each of several resident surgeons operated upon one patient of this series. Most surgeons agree that divided tendons are more difficult to repair in the hand than in other parts of the extremity. In terms of this factor alone, the present results are satisfactory, but they can and should be improved.

REFERENCES

1. Altemeier, W. A.: Treatment of Fresh Traumatic Wounds. J. Am. Med. Assn., **124**: 405–408, 1944.
2. Altemeier, W. A.: Personal Communication.
3. Bunnell, Sterling: Repair of Tendons in the Fingers and Description of Two New Instruments. Surg., Gynec., and Obstet., **26**: 103–110, 1918.
4. Garlock, J. H.: Repair of Wounds of the Flexor Tendons of the Hand. Ann. Surg., **83**: 111–122, 1926.
5. Koch, S. L.: The Treatment of Open Wounds. Bull. Am. Coll. Surgeons, **25**: 176–178, 1940.
6. Koch, S. L.: Injuries of the Parietes and Extremities. Surg., Gynec., and Obstet., **76**: 1–22, 189–196, 1943.
7. Koch, S. L., and Mason, M. L.: Division of the Nerves and Tendons of the Hand. With a Discussion of the Surgical Treatment and Its Results. Surg., Gynec., and Obstet., **56**: 1–39, 1933.

8. Koch, S. L., and Mason, M. L.: Purposeful Splinting Following Injuries of the Hand. Surg., Gynec., and Obstet., **68**: 1–16, 1939.
9. Mason, M. L.: Rupture of Tendons of the Hand. With a Study of the Extensor Tendon Insertions in the Fingers. Surg., Gynec., and Obstet., **50**: 611–624, 1930.
10. Mason, M. L.: The Surgical Principles Involved in the Treatment of Open Injuries. Western J. Surg., **45**: 239–248, 1937.
11. Mason, M. L.: The Immediate Treatment of Open Wounds of the Hand. Mississippi Valley Med. J., **61**: 136–143, 1939.
12. Mason, M. L.: Primary and Secondary Tendon Suture. A Discussion of the Significance of Technique in Tendon Surgery. Surg., Gynec., and Obstet., **70**: 392–402, 1940.
13. Mason, M. L., and Allen, H. S.: The Rate of Healing of Tendons. An Experimental Study of Tensile Strength. Ann. Surg., **113**: 424–456, 1941.
14. Miller, Harry: Repair of Severed Tendons of the Hand and Wrist. Statistical Analysis of 300 Cases. Surg., Gynec., and Obstet., **75**: 693–698, 1942.
15. O'Shea, M. C.: The Treatment and Results of 870 Severed Tendons and 57 Severed Nerves of the Hand and Forearm. Am. J. Surg., **43**: 346–366, 1939.
16. Siler, V. E.: Management of Injuries and Infections of the Upper Extremities. J. Am. Med. Assn., **124**: 408–412, 1944.
17. Winfield, J. M.: Anatomic Diagnosis of Injuries of the Hand. J. Am. Med. Assn., **116**: 1367–1370, 1941.

DISCUSSION

Transplantation of the External Oblique Muscle

(*Continued from page 217*)

ber of muscles, perhaps of all the muscles, supporting and moving the hip joint. Apparently, however, the abductors and the extensors provide the major part of the stabilizing action. The tensor fasciae latae, the glutaeus minimus, and the anterior portion of the glutaeus medius are the prime abductors of the hip. The posterior part of the glutaeus medius and the glutaeus maximus are the prime extensors of the hip. This arrangement would indicate a close association between abduction and extension in providing hip stability.

In our Clinics, we have had the most satisfactory results when the origin of a strong tensor fasciae latae was transplanted backward into the vicinity of the posterior superior spine. We, like the authors, have had some very gratifying results and some very disappointing ones. After reading this contribution, I am inclined to consider combining the two operations—transference of the external oblique with posterior transference of the origin of the tensor fasciae latae—as indeed was suggested in this paper, feeling that the combined operation may give sufficient power to ensure more satisfactory stabilization.

It should be emphasized that in this operation, as in others which have been reported, a careful selection of cases is of paramount importance, if satisfactory results are to be gained and useless surgery avoided. A strongly functioning muscle must be available, and reasonably well-functioning supporting muscles must be present before muscle transference can be successful, whether the external oblique, the tensor fasciae latae, or the erector spinae is to provide the stabilizing power.

We are indebted to the authors of this paper for reporting the results of an operation which should be useful in the treatment of instability of the paralytic hip.

Dr. Leo Mayer, New York, N. Y.: In treating abductor limp, I, like Frank Ober, have attempted to utilize the erector spinae group as a substitute for the weak or paralyzed abductors. My first patient, operated upon twenty years ago and now grown to womanhood, has definitely less abductor limp than before her operation, and is able, when resting on her side, to hold her lower extremity abducted against gravity. There are, however, despite that fairly good result, only about half a dozen cases in this group. In another series, I employed the Telson operation,—that is, the forward shift of the glutaeus maximus; and in a third group, the Dickson method of posterior shifting of the tensor fasciae latae. Dissatisfied with the results in all three groups, I tried to utilize the external oblique muscle in one case, but failed because I did not free a large enough muscle flap.

The authors are to be congratulated. Their results are the best I have seen thus far, even though they are not perfect. By performing their operation in conjunction with transplantation of the erector spinae, we may be able to construct an efficient substitute for the powerful abductor group of muscles.

Their operation requires careful preoperative study, so as to select suitable cases, an accomplished surgical technique, and painstaking after-treatment.

ADDITIONAL ELASTIC HAND SPLINTS

BY STERLING BUNNELL, M.D., AND LOT D. HOWARD, JR., M.D., SAN FRANCISCO, CALIFORNIA

The principle of using elastic or spring splints to exercise, mobilize, or change the position of stiffened joints in injured hands has been eminently successful. They coax joints into the position of function or complete the muscle balance in paralysis [1]. Accordingly, the splints described here have been devised recently.

The object of the first of these splints or adaptations is to change the hand from the position of non-function (in this case with the wrist flexed and the fingers extended) into the position of function. This is done by three attachments which increase the use of the knuckle-bender splint. Two of the attachments fit onto the splint by two wire rods, thrust into metal sleeves.

The distal attachment to flex the middle finger joints is an additional flat, padded segment, hinged to the distal segment of the knuckle-bender splint. Both attachment and splint are powered by rubber bands (Figs. 1-A and 1-B).

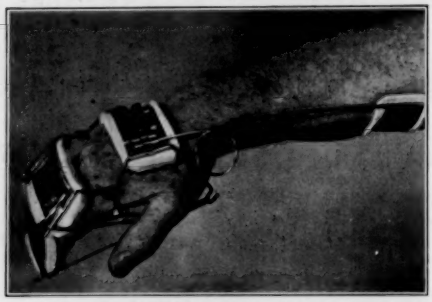

FIG. 1-A

FIG. 1-B

Splints to change position of hand from that of non-function, with fingers in extension, to that of function.

Fig. 1-A: A combination of the knuckle-bender splint and the Oppenheimer splint to flex the proximal finger joints and dorsiflex the wrist, with an extra segment of the knuckle-bender splint to flex the middle finger joints. Position of non-function is shown.

Fig. 1-B: Splint has drawn hand into position of function.

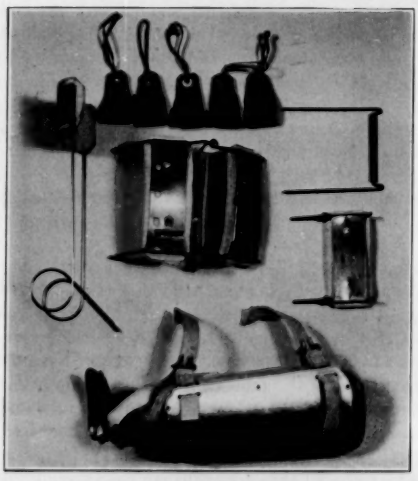

FIG. 1-C

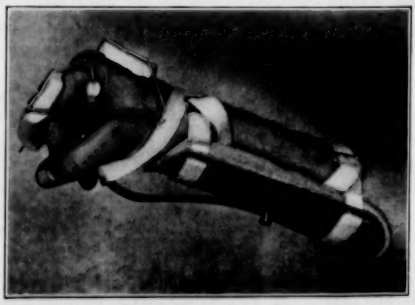

FIG. 1-D

Fig. 1-C: Various attachments for knuckle-bender splint to dorsiflex the wrist, to flex the proximal and middle finger joints, and to draw out the middle and distal finger joints into extension.

Fig. 1-D: Combination of knuckle-bender splint to flex the proximal finger joints and spring cock-up splint to dorsiflex the wrist. Attachments can be added to this to flex or extend the fingers.

FIG. 2-A

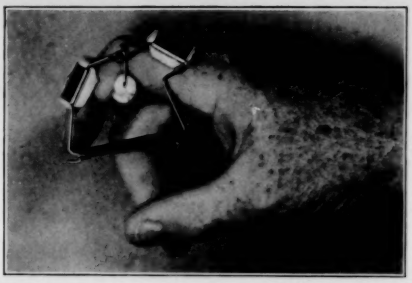

FIG. 2-B

Miniature knuckle-bender splint to flex middle (or distal) joint of finger.
Fig. 2-A: Finger is stiff in extension.
Fig. 2-B: Middle joint is being gradually flexed by splint.

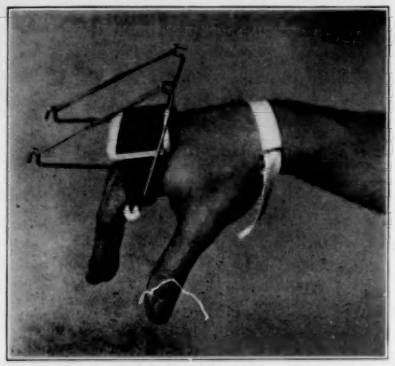

FIG. 3-A

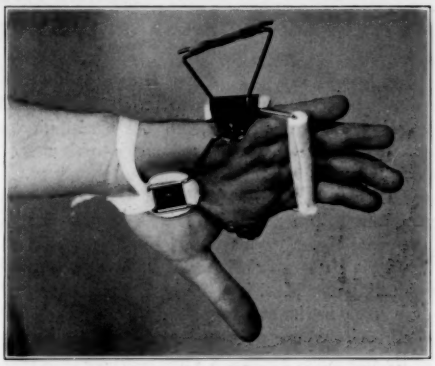

FIG. 3-B

Reverse knuckle-bender splint to extend the proximal finger joints gradually, in a case of mild contracture of the intrinsic muscles.
Fig. 3-A: Shows the position of deformity.
Fig. 3-B: Proximal finger joints have been drawn into extension by the three-point elastic pressure of the splint.

The purpose of the other two attachments is, by gentle elastic persuasion, to force the wrist into dorsiflexion. One is by use of a splint of the Oppenheimer type, in which the distal two wires fit into sleeves for attachment (Figs. 1-A and 1-B); the other is by using a spring cock-up splint, the hand piece of which is notched to fit over the cross piece of the knuckle-bender splint (Figs. 1-C and 1-D). Either may be used alone with the knuckle-bender, or may be combined with the attachment to flex the fingers (Fig. 1-B) or with the attachment to straighten them, as previously described [1].

For a single stiffened finger joint, a miniature replica of the knuckle-bender splint can be worn, the rest of the hand being left unencumbered. The elastic tension is maintained by light-weight druggist's bands (Figs. 2-A and 2-B). The angulation of the proximal wire extension is intended to accommodate for the soft tissues of the finger webs.

The purpose of the third splint is to draw out the flexion contracture of the proximal finger joints, due to ischaemic fibrosis of the muscles of the hand. The treatment of these hands presents a difficult problem. While yielding temporarily to splinting, they usually require operative intervention, only the less severe cases being benefited by the long period of splinting. Splints applying elastic extension to the ends of the fingers serve only to accentuate the existing hyperextension of the middle and distal joints of the finger.

To give positive elastic extension to the proximal segments of the fingers only, thus overcoming the contracture of the proximal finger joints, a "reverse knuckle-bender"

splint was designed (Figs. 3-A and 3-B). It consists of a base plate, stabilized in the palm by a web strap fastened about the wrist. Counterpressure, applied over the proximal joints by a slightly curved, felt-padded plate, allows elastic pressure to be brought to bear on the volar aspect of the proximal finger segments, near their distal ends, by a padded bar. The extended stiff-wire outriggers provide good mechanical advantage and a convenient way to apply the number of elastic bands necessary.

1. Bunnell, Sterling: Active Splinting of the Hand. J. Bone and Joint Surg., **28**: 732–736, Oct. 1946.

ROBERT VIVIAN FUNSTEN

1892–1949

Dr. Robert Vivian Funsten, known to all as Bob, died suddenly on July 25, 1949. Having weathered, apparently successfully, a severe illness in February, he seemed to have returned to his usual jovial self by late spring and early summer, and took his familiar place in the activities at Colorado Springs. The news of his untimely death came as a great shock to his many friends. Bob will be sorely missed by all who knew him, especially by his colleagues in orthopaedic surgery.

Dr. Funsten was born in Portsmouth, Virginia, on Christmas Day, 1892; his parents were the late Rev. James Bowen Funsten, D.D., L.L.D., Episcopal Bishop of Idaho, and the late Ida Vivian Pratt Funsten. He received his early education in the Episcopal High School, Alexandria, Virginia (1907 to 1910), and then entered the University of Virginia, where he studied in the College and the Departments of Mechanical Engineering and Medicine, receiving the degree of Doctor of Medicine in 1918. He was active in many student organizations and was a member of the Alpha Tau Omega, Phi Rho Sigma, and Lamda Pi fraternities, several social groups, and the honorary fraternity of Alpha Omega Alpha.

He was commissioned a First Lieutenant in the Medical Corps in 1918, and served with Mobile Hospital No. 102.

After the first World War, Dr. Funsten went to the University of Iowa, where he worked as Assistant Professor of Orthopaedic Surgery under Dr. Arthur Steindler from 1919 to 1922; there his mechanical talents were appreciated and stimulated.

From 1922 to 1932, Dr. Funsten lived in Detroit and practised his specialty in association with Dr. Frederick C. Kidner. In 1932 he became Professor of Orthopaedic Surgery at the University of Virginia, which position he held until he died. While at the University of Virginia, he expanded the service in the University Hospital and established a convalescent home for crippled children, which allowed many more children to benefit from his talents in surgery and his genial, kindly spirit. As a teacher, he was interesting and stimulating to students and, at the same time, he encouraged and trained a number of orthopaedic surgeons who are now established in various communities.

Dr. Funsten was a member of the American Medical Association; The American Orthopaedic Association; the Clinical Orthopaedic Society; Virginia and Albemarle County (Virginia) Societies; Wayne County (Michigan) Medical Association; Detroit Academy of Surgeons and Medical Club (President in 1929); and Fellow of the International Academy of Medicine.

He made many contributions to orthopaedic surgery in the way of papers and mechanical devices, which have added much to the specialty; his textbook on orthopaedic nursing is regarded by many as the best in its field.

Dr. Funsten is survived by his wife, Marguerite Churchill Funsten, and four children, Dorothy Funsten Craig, Patricia Anne Funsten Cady, James Churchill Funsten, and Robert Vivian Funsten, Jr.

It is with deep regret that we record the untimely death of Professor T. P. McMurray of Liverpool, President-Elect of the British Medical Association, and formerly President of The British Orthopaedic Association. His contributions to the advancement of orthopaedic surgery have been substantial.

News Notes

The American Orthopaedic Association will hold its Annual Meeting at Virginia Beach, Virginia, May 23, 24, 25, and 26, 1950.

The Annual Meeting of the **American Academy for Cerebral Palsy** will be held at the Waldorf-Astoria Hotel, New York City, on Friday and Saturday, February 17 and 18. The scientific sessions will be open to visiting orthopaedic surgeons and any physicians interested in the problem of cerebral palsy. These sessions are scheduled for Friday afternoon, February 17, from two to five o'clock and Saturday morning, February 18, from nine to twelve. All phases of cerebral palsy will be discussed.

The Secretary of the organization is Dr. Meyer A. Perlstein, 4743 North Drake Avenue, Chicago 25, Illinois.

THE AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS

The Seventeenth Annual Meeting of The American Academy of Orthopaedic Surgeons will be held at the Waldorf-Astoria, New York City, February 11 to 16, 1950, under the presidency of Dr. Mather Cleveland.

Registration will begin at nine o'clock, Saturday morning, February 11. The scientific and technical exhibits will be ready for inspection at that time. The Audio-Visual Program will be presented Saturday afternoon and Sunday morning.

An interesting group of Instructional Courses has been arranged by the Instructional Course Committee. These courses will be presented Sunday afternoon and Monday morning. At the Instructional Course Dinner on Monday evening, an "Information Please" program will be given, at which time unusual and interesting cases will be presented to "stump the experts".

The first session of the Scientific Program will be held on Monday afternoon. The Scientific sessions will continue through Tuesday, Wednesday, and until noon on Thursday. A most interesting Scientific Program has been prepared by the Program Committee.

New members of the Academy will receive their certificates on Monday afternoon. The Presidential Address will be given on Tuesday morning. Tuesday evening will be open for alumni and hospital dinners.

The annual banquet of the Academy will be held on Wednesday evening, at which time the Kappa Delta and other awards will be made.

The **American Society for Surgery of the Hand** will hold its Annual Meeting in New York City, February 10 and 11. The sessions on Friday, February 10, will be held at the New York Academy of Medicine. The following program has been planned:

Morning Session (9:30 A.M.)

Safety Factors in Tourniquet Hemostasis. Julian M. Bruner, M.D., Des Moines, Iowa
Tumors of the Hand. Michael L. Mason, M.D., Chicago; Henry C. Marble, M.D., Boston; and Arthur Purdy Stout, M.D., New York City
The Motor Branch of the Ulnar Nerve in the Palm. Joseph H. Boyes, M.D., Los Angeles
Dynamic Splinting. Sterling Bunnell, M.D., San Francisco
Brachial Block Anaesthesia with Xylocain. Erik Moberg, M.D., Goteborg, Sweden

Afternoon Session (1:30 P.M.)

Phylogeny of the Hand. William K. Gregory, Ph.D., D.Sc., New York City
Early Development of the Hand and Its Relation to Function. Emanuel Kaplan, M.D., New York City
Congenital Anomalies of the Hand. Arthur J. Barsky, M.D., New York City
(a) Joint Arthrodesis to Increase Hand Function. (b) Antecubital Donor Skin Grafts. William H. Frackelton, M.D., Milwaukee, Wisconsin
An Experimental Study of the Tensile Strength of Tendon Repair. Raymond M. Curtis, M.D., Baltimore
Subcutaneous Fasciotomy in Dupuytren's Contracture. Vernon Luck, M.D., Los Angeles

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The **Sociedad Mexicana de Ortopedia** is planning to hold its first convention of orthopaedic surgeons during this year. The officers of the Society are: *President*, Pedro Rosas, M.D.; *Secretary*, Rafael Farrera Rojas, M.D.; and *Treasurer*, Max Luft, M.D.

The Journal is happy to announce the recent founding in Caracas of the **Sociedad Venezolana de Cirugia Ortopédica y Traumatología.** The organization is made up for the most part of surgeons whose work is limited to this branch of surgery.

The Board of Directors of the new Society are: *Chairman*, Dr. Andres Gutierez Solis; *Vice-Chairman*, Dr. A. Pietri, hijo; *Executive Secretary*, Dr. Jorge Figarella; *Treasurer*, Dr. Juan G. Yanez; *Librarian*, Dr. Cesar Acosta; and assistant members, Dr. Armando Parada and José Antonio Villegas.

Dr. G. W. N. Eggers, Professor of Orthopaedic Surgery at the University of Texas, Medical Branch, was awarded the **Robert Danis Prize** at the meeting of the **International Society of Surgery,** held in New Orleans in October. This prize, established by the friends of Robert Danis, formerly Professor of Surgery at the University of Brussels, is awarded for important work on the operative treatment of fractures. It was presented to Dr. Eggers on the basis of his studies on the "Internal Contact Splint" and "The Influence of the Contact-Compression Factor on Osteogenesis in Surgical Fractures". Both of these articles were published in *The Journal*, the former in the issue of January 1948, and the latter in the issue of October 1949.

The Fourth Annual Meeting of the **New Jersey Orthopaedic Society** was held on October 8, 1949 at the Alfred I. duPont Institute in Wilmington, Delaware. The following program was presented:

Morning Session

Introductory Remarks: A. R. Shands, Jr., M.D.
Bacteriological Aspects of Orthopaedic Surgery: Armine T. Wilson, M.D.
Biochemical Aspects of Orthopaedic Surgery: Paul B. Hamilton, M.D.
Treatment of Club-Foot: David J. King, M.D.
Recent Visits to Orthopaedic Clinics in Europe: S. W. Casscells, M.D.
Presentation of Cases: Staff of the A. I. duPont Institute.

Afternoon Session

Mechanics of Bone and Joint Surgery: Mr. J. Collison.
Congenital Hips: Follow-up of Open Reduction: Paul J. Strassburger, Jr., M.D.
Case Reports: Myosarcoma. Septic Arthritis: Keith E. Haines, M.D.
Tibial Plateau Fractures: Paul Finegan, M.D.

The National Foundation for Infantile Paralysis, Inc. announces that it is offering post-graduate research fellowships for one to three years in the fields of virology, orthopaedic surgery, pediatrics, epidemiology, and neurology. These fellowships are available to properly qualified candidates whose objectives are research and teaching in these fields. Eligibility requirements include United States citizenship, sound health, degree of Doctor of Medicine or Doctor of Philosophy, two years' residency training in the specialty, and a program of study and detailed plan of investigation.

Further information concerning the fellowship programs may be obtained from the Professional Educational Division of the National Foundation for Infantile Paralysis, Inc., 120 Broadway, New York 5, N. Y.

Columbia University announces an extension and broadening of its program for Graduate Training in Physical Medicine and Rehabilitation at the **Columbia-Presbyterian Medical Center** and the **Institute for the Crippled and Disabled.** A three-year period of broad and intensive training is available to young doctors of high medical caliber, who have had at least one year of internship and plan to acquire training in physical medicine and rehabilitation.

The program will start with a brief didactic introduction in the techniques and philosophy of physical medicine and rehabilitation, followed by three phases of training:

(a) Clinical experience and responsibility in the practice of physical medicine in the several departments of the Medical Center, and in rehabilitation at the Institute for the Crippled and Disabled.

(b) Periods of clinical training under the Departments of Neurology and Orthopaedic Surgery, and in special fields of internal medicine, to supplement the student's general experience in those clinical fields where physical medicine plays the greatest role.

(c) Opportunities for research or instruction in the basic sciences, according to the student's interest and aptitude.

Further information may be obtained from Associate Professor Robert C. Darling, Coordinator of Physical Medicine and Rehabilitation, College of Physicians and Surgeons, 630 West 168 Street, New York 32, N. Y.

Book Reviews

ATLAS OF PERIPHERAL NERVE INJURIES. William R. Lyons, Ph.D., and Barnes Woodhall, M.D. Philadelphia, W. B. Saunders Company, 1949. $16.00.

Dr. Lyons, neuro-anatomist, and Dr. Woodhall, neurosurgeon, have assembled from their wartime experience a sweeping panorama of the gross and histological appearance of damaged nerves. In addition, they have presented a convenient summary of suggestions for treatment, based on their own studies and on the literature. The *Atlas* is profusely complete, and the quality of the reproductions is so good that the reviewer can concur in the authors' recommendation that the pictures be examined with a hand lens. Each group of photographs of a lesion is accompanied by enough clinical data to clothe the illustration with the realities which accompanied it. The well selected bibliography of 190 references with full titles includes most of the data thus far available in English from World War II, as well as important earlier articles. An omission is the superb four-volume work of Förster, published ten years after World War I as a series of supplements to the *Handbuch der Neurologie.*

As one of their major recommendations, the authors urge that the definitive repair of traumatized nerves be deferred usually until after the third week following injury, and that it be done after healing of the original wound. They point out that, if the nerve is repaired earlier, the epineurium is too thin for easy suture, mobilization of the nerve to obtain necessary length may spread infection, and the extent of the damaged area of the nerve cannot be recognized by the surgeon. On the other hand, if the suture is deferred beyond one or two months, Schwann tubules in the distal nerve segment will have begun to shrink in size and to be surrounded by fibrosis, which will render incomplete such regeneration as occurs. The validity of this advice will be tested by the actual late results being recorded in the massive series of nerve injuries now under study at several centers in this country.

Two of the authors' main findings with respect to the vexing question of the amount of resection needed at the proximal ends of the nerves were: (1) the effects of battle trauma to nerves extend farther central to the main site of injury than is apparent to the eye, but (2) provided the nerve is resected proximal to most of the gross pathological change, it is of no great importance to reach the level of entirely normal nerve fibers. Microscopic study usually shows degenerative changes in the fibers for about one centimeter proximal to the last point of alteration grossly detectable.

The writers have a particularly instructive section on traction lesions. The illustrations prove that the pathological changes in these conditions can be more severe than in any other form of nerve injury, "yet in practice such cases are most likely to suffer from a policy of non-intervention and procrastination".

Their section on associated nerve and vascular lesions contains some striking facts. A major vascular injury occurred in 13 per cent. of the patients with peripheral-nerve suture reported in the Peripheral Nerve Registry. Many vascular lesions, notably the small arterial aneurysms, may be unrecognized prior to operation and found without warning at a routine exploration of a nerve. Hence the surgeon engaging in the latter procedure should also have a full knowledge of vascular surgery.

It is a pleasure to congratulate the authors on providing a unique reference work which, one fears, will become even more widely needed in the not-distant future than at present.

NEOPLASMS OF BONE AND RELATED CONDITIONS. THEIR ETIOLOGY, PATHOGENESIS, DIAGNOSIS, AND TREATMENT. Bradley L. Coley, M.D. New York, Paul B. Hoeber, Inc., 1949. $17.50.

Because of Dr. Coley's long association as Attending Surgeon in the Bone Tumor Department of the Memorial Hospital for Cancer and Allied Diseases in New York, an uncommonly large number of bone neoplasms have passed under his scrutiny. To the clinical aspects of such diseases the author has devoted most of his attention in this book, and for that reason it will be most welcome to the general physician who does not often encounter such lesions. Problems of diagnosis are fully discussed, as well as various therapeutic measures and their success or failure. Section One is devoted to the classification, etiology, and diagnosis of neoplasms of bone; a second section is given over to benign tumors and tumor-like lesions. Other sections deal with problems of surgical treatment and radiation therapy. A chapter on metastatic tumors involving bones will be of great interest. There are even a few pages explaining briefly what has been accomplished in the experimental production of bone sarcoma.

All in all, Dr. Coley has here compiled practically the entire mass of available material in this broad field. It is as though, through the pages of this comprehensive study, the reader were given a digest of the material in the files of the Bone Sarcoma Registry.

ATLAS OF HUMAN ANATOMY. DESCRIPTIVE AND REGIONAL. Vol. I. Osteology, Arthrology, Myology. M. W. Woerdeman, M.D., F.R.N.A.Sc. Amsterdam, Wetenschappelijke Uitgeverij, Baltimore, The Williams and Wilkins Company, 1948. $12.50.

Because medical students in many countries have insufficient anatomical material for dissection, Dr.

Woerdeman, who is Professor of Anatomy and Embryology at the University of Amsterdam, is preparing an atlas of anatomy in which each plate is intended to be a true representation of an individual specimen, rather than a composite view of more than one dissection. He hopes that, by comparison of plates and of actual dissections, the student will be made aware of the individual variations which exist in the structure of the human body.

This work is to be presented in three volumes. Volume I deals with the locomotor apparatus. The later volumes will be concerned with splanchnology, the circulatory apparatus, nervous system and sense organs, and the peripheral vessels and nerves.

The illustrations are of exceptionally good quality. Many plates are full-page; a few are in color. The section on myology is of especial interest, in that the muscle position and attachments, shown in color, are superimposed on the bone structure. The nomenclature is in Latin and follows the *Basle Nomina Anatomica*, brief captions being given in English. No text is included.

This volume is beautifully prepared and should be of value to practitioners as well as students.

TUMORS OF BONE. Ed. 3. Charles F. Geschickter, M.D., and Murray M. Copeland, M.D. Philadelphia, J. B. Lippincott Company, 1949. $17.50.

This third edition of one of the most outstanding studies of tumors of bone to be published presents an even more complete review of all of the available material on this subject. In the original work, published in 1931, the writers presented a storehouse of information, containing not only descriptions of the lesions then known, but also suggestions for classification of tumors and a valuable discussion of differential diagnosis. The present edition has been increased by 100 pages over the second edition, which appeared in 1937; and, because of the passage of time, the writers have been able to complete follow-up reports, with results which they feel justify their original classifications in regard to benign giant-cell tumor, chondroblastoma, chondrosarcoma, and other entities.

New chapters are included on fibrous dysplasia, osteoid osteoma, tumors of the spine, endocrine changes in bone, and other rare diseases of bone. There has been added a whole new section on tumors of non-osseous origin, and the problems arising from skeletal metastases are taken up. The chapter discussing endocrine disturbances and some rare diseases of unknown etiology will be of primary interest, in view of the current importance of all phases of endocrinology.

One of the most valuable features of this third edition is the excellent and useful format of each chapter. Wherever possible, the following plan is adhered to: first the name of the neoplasm to be discussed, then its clinical features, roentgenographic features, gross pathology, microscopic features, histogenesis, etiology, treatment and prognosis, and finally a summarizing section.

The illustrations of roentgenograms, photomicrographs, and gross specimens are of superior quality, and their number has been increased. The Table of Contents has been widened in its scope and would seem to be of greatly increased usefulness. This also applies to the Index. As with the previous editions, at the end of each chapter there is a bibliography.

SKIN GRAFTING. Ed. 2. James Barrett Brown, M.D., and Frank McDowell, M.D. Philadelphia, J. B. Lippincott Company, 1949. $7.50.

Brown and McDowell, in the second edition of their monograph on "Skin Grafting", have again made an outstanding contribution to the literature on the surgery of repair. The material is presented in the simple, straightforward manner which is characteristic of other publications by these authors. The emphasis is on broad principles applicable to all phases of reparative surgery, and yet the technical details of wound management, skin transplantation, and preoperative and postoperative care are clearly and concisely recorded, so that they are understandable to surgeons with little experience in the field.

The material from the first edition, which was concerned principally with the early and late care of burns, has been thoroughly revised. The discussion of burns is detailed "because burns present an unusually good opportunity for the study of wound healing". In addition to these valuable revisions, the scope of the book has been expanded to include recent advances, some of which stem from Dr. Brown's war experiences. Full-thickness grafts from the base of the neck, composite grafts of skin and cartilage from the ear, permanent pigment injection, ear and nose reconstruction, and repair for a variety of defects resulting from removal of tumors, mechanical injuries, and chemical and electrical burns are described.

There is a splendid section on the pathology and treatment of radiation injuries from ill-advised exposure to roentgen rays and from atomic radiation in which excessive local exposure in an interval of a few seconds greatly accelerates the destructive processes. Dr. Brown has had experience with the latter as Consultant to the Atomic Energy Commission.

To the surgeon dealing with burns and other trauma, the chapter on "Skin Grafting in Military Plastic Surgery" will prove invaluable. World War II stimulated an increasing awareness of the importance of the surface repair, either as a primary definitive measure or as a preparation for later deep surgery. Many principles and methods evolved during that period are described and illustrated.

The philosophy of patient care, so well expressed on page 290 and in the concluding paragraphs of the last chapter, reflects the high-mindedness and character of the authors, and is a lesson to all whose responsibility it is to carry patients through a long period of surgical rehabilitation.

PNEUMOARTHROGRAPHY OF THE KNEE JOINT WITH PARTICULAR REFERENCE TO THE SEMILUNAR CARTILAGES. Kjeld Andersen. *Acta Orthopaedica Scandinavica*, Supplementum IV, 1948.

Kjeld Andersen, while assistant radiologist at the Copenhagen County Hospital in Gentofte, carried out pneumo-arthrographic studies of the knee joint. His material is presented in an orderly manner, with an introduction of historical interest.

The pneumo-arthrographic studies were made with particular reference to the semilunar cartilages, and close attention to detail is emphasized in the injection of atmospheric air and in roentgenographic technique. Normal knees were thoroughly studied by means of anterior-posterior, lateral, and special-view roentgenograms. Roentgenograms were also taken of disc-shaped frozen sections of preparations of the knee, including sections of the tibia and the femur which were cut vertically and then radially through the extended knee; these were used as a base-line for the normal.

Seven cases of error in preoperative diagnosis were made by the method, and these cases are presented in detail. The material studied comprises 150 cases.

This monograph is an excellent review of the subject. Its usefulness is limited, however, and it does not replace the findings of a good physical examination or add much to the usual roentgenograms, except as a refinement aid in diagnosis.

L'ORGANISATION DES Os. P. Lacroix. Liège, Éditions Desoer, 1949.

This study of osteogenesis is based on research undertaken by the author to clarify certain questions of surgical pathology. It deals with the structure of long bones of the adult, the method of their growth and development; the growth of periosteum and its role in the organization of the diaphysis; the role of the epiphyseal cartilage in the growth in length of bone, and its function; and the growth of the bone marrow.

The book presents an important study in a neglected field, and should be of value to all who in any capacity are interested in the question of the formation and structure of bone.

The bibliography included will be helpful to all working in this field.

This book is a compilation of articles prepared by Dr. Lacroix, two of which have been published in the *Journal*. The first of these articles, "Organizers and the Growth of Bone", appeared in the issue of April 1947; the second, "The Growth of the Bone Marrow", in October 1949.

MUSCLES. TESTING AND FUNCTION. Henry O. Kendall and Florence P. Kendall. Baltimore, The Williams and Wilkins Company, 1949. $7.50.

In these days, when the orthopaedic surgeon interested in the treatment of poliomyelitis is subjected to a barrage of treatises dealing with antispasmodics, ranging from curare to tolserol, all stemming from the Kenny emphasis on "spasm" as the all-important element of infantile paralysis, it is refreshing to read a book which stresses first principles and presents so clearly that most significant factor in the treatment and prognosis of the paralyzed patient,—the exact determination of muscle power. This book is a model of accuracy, thoroughness, and clarity of presentation. In a series of photographs, the action of every important muscle is clearly shown. The text explains how the tests are performed and what errors can be avoided. The two opening chapters on fundamental principles of muscle testing and on charts should be studied by every orthopaedic student.

This book is an important addition to our armamentarium in the fight against poliomyelitis, and should be part of the working equipment of every physician who wants to treat paralytic cases with scientific precision.

COMROE'S ARTHRITIS AND ALLIED CONDITIONS. Ed. 4. Joseph L. Hollander, M.D., Editor. Philadelphia, Lea and Febiger, 1949. $16.00.

The first edition of Comroe's *Arthritis* appeared in 1940, and was soon followed by a second and, in 1944, a third edition. The present fourth edition represents a revision by seventeen collaborating editors under the leadership of Joseph L. Hollander. While the editorial board is composed mainly of internists with a special interest in the rheumatic diseases, two orthopaedic surgeons, John G. Kuhns and Theodore A. Potter, have been responsible for the chapters on the orthopaedic management of arthritis and on painful feet.

Notwithstanding the advances in our knowledge of joint disease and the inclusion of new material, the editors deserve commendation for their achievement in shortening the present text by over 200 pages. When

the book is read as a whole, still further condensation seems possible and desirable by the use of cross references to avoid repetition of material on identical subjects. To cite one example, Bennett's work on the shoulder lesions in baseball pitchers is summarized in the chapter on "The Painful Shoulder" as well as in that on "Traumatic Arthritis".

Additional brevity and clarity might also be obtained by a more orderly and concise presentation of subjects covered in certain individual chapters, and by dispensing with figures which fail to illustrate the adjacent text. A minor, but important, criticism can be laid to the proofreading.

These criticisms may be regarded as constructive and are not meant to detract from the reviewer's high regard for this text, which may justly be considered the most useful on the subject of joint disease yet to appear in this country. Many of the chapters represent scholarly, well-organized appraisals of our knowledge in certain fields of arthritis, which cannot be duplicated under one heading elsewhere. The summaries in box form, which are frequently interspersed throughout the text, provide valuable, quickly-read résumés of important material. Hench and Kendall's work with cortisone and ACTH was announced just before the book went to press and is included in brief summary, with the perhaps premature implication that rheumatoid arthritis is thus a disease of endocrine etiology. The subject of therapy, on which most publications on arthritis stand or fall, is in general well handled, with certain exceptions, such as the interval management of gout and the treatment of backache, where a more positive and critical stand would have been helpful to the reader.

Subsequent editions should establish this book even more firmly as an authoritative treatise on arthritis, which can be recommended to both internists and orthopaedic surgeons.

A Textbook of Surgery. Ed. 5. By American Authors. Frederick Christopher, B.S., M.D., F.A.C.S., Editor. Philadelphia, W. B. Saunders Company, 1949. $13.00.

The fifth edition of this work, which Dr. Christopher defines as "a cross-sectional presentation of the best in American surgery", has been prepared by the collaborative effort of almost two hundred well-known surgeons. In so far as possible, the material has been set up according to etiology, pathology, diagnosis, prognosis, and treatment. As in previous editions, the plan has been to present only accepted methods of surgery, omitting that which is controversial or insufficiently tested, so that the student may have an authoritative source of information.

In this edition new sections have been added, the arrangement of material has been altered somewhat, and many portions have been re-written. Extensive changes, of interest to the orthopaedic surgeon, concern chemotherapy of surgical infections, pathology and repair of fractures and their operative treatment, fractures of the femur, and aseptic surgical technique.

To those familiar with earlier editions of this textbook, it needs no recommendation. The high standards set by Dr. Christopher in the past have been well maintained in the most recent edition.

The Science and Art of Joint Manipulation. Vol. 1, Ed. 2—The Extremities. James Mennell, M.A., M.D., B.C. (Cantab.). London, J. and A. Churchill, Ltd., 24 shillings; Philadelphia, The Blakiston Company, 1949. $7.50.

This second edition of Dr. Mennell's contribution to the science of joint manipulation appears to offer all of the excellent material found in the first edition, plus some valuable new discussions. Three new and two alternative techniques are described,—two concerning the manipulation of the shoulder joint and one that of the cuboid joints. The number of illustrations has been increased, and several of the roentgenograms have been replaced.

The especial value of this volume would seem to lie in its excellent organization and clarity, making it useful not only to the experienced orthopaedic surgeon, but also to the physical therapist and the student. Dr. Mennell's aim has been more than the mere setting forth of the bare scientific facts relating to joint manipulation. He has clarified the successful application of these facts.

Surgical and Maxillofacial Prosthesis. Oscar Edward Beder. New York, King's Crown Press, 1949. $3.00.

The importance of prosthetic restoration in the correction of non-operable maxillofacial defects is now well recognized. Prostheses have also been used as temporary aids in surgical operations by immobilizing parts and giving support to soft tissues.

The book describes the technique of the construction of various prosthetic appliances. It is divided into seven chapters, describing the construction of splints and obturators, prosthetic restoration of various parts of the face and jaws, and the use of dental composition. The fourth chapter, devoted to the construction of protective shields for use in oral and face radiation therapy, is of especial interest.

Primarily, the book is a laboratory manual for dental technicians.